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MASTITIS OF THE COW

BY

SVEN WALL

ASSISTANT IN THE VETERINARY HIGH SCHOOL AT STOCKHOLM

WITH 29 ILLUSTRATIONS

AUTHORIZED TRANSLATION WITH ANNOTATIONS

BY

DR. WALTER J. CROCKER. B.S., V.M.D.

PROFESSOR OF VETERINARY PATHOLOGY, UNIVERSITY OF PENNSYLVANIA

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TRANSLATOR'S PREFACE

THE dearth of detailed scientific and practical literature on the subject of inflammation of the udder in cows prompted me to prepare a translation of Professor Sven Wall's excellent book, "Die Euterentzundungen der Kuh."

Not only is the subject adequately treated in all its important practical relationships, but the conclusions drawn have been established upon sound principles of pathology and by decisive animal experimentation. The tubulo-alveolar system, Professor Wall observes, is lined by epithelium, cubical and flat, which constitutes the *parenchymatous* tissue. This is supported by interstitial connective tissue which constitutes the *stroma.*

Bacterial invasion of the interstitial connective tissue comprises a *stroma-infection,* the presence of which is manifested, more or less obviously, by a reaction on the part of the body to the bacterial irritation called an inflammation or *interstitial mastitis.* This may be primary or secondary depending upon the manner of its inception, or acute or chronic depending upon its duration and the character of the specific etiological factors.

Bacterial invasion of the tubulo-alveolar system or parenchymatous passages, and thus necessarily the milk which is contained in them, he calls a *parenchyma-infection* or *milk-infection,* the presence of which is manifested by a reaction on the part of the body to the bacterial irritation called an inflammation or *parenchymatous mastitis.* This may be primary or secondary depending upon the manner of inception, or acute or chronic depending upon its duration,—and based upon the character of its inflammatory exudate it is classified as *catarrhal mastitis, sero-hemor-*

rhagic mastitis, croupous mastitis, purulent mastitis, or *croupo-purulent mastitis.* These terms indicate the presence of an inflammatory process of the udder in which the *parenchyma is affected with various degrees of severity, which are distinguished by the character of their exudates.*

In the acute form of these types of parenchymatous mastitis, *except the catarrhal type,* the udder becomes swollen, hot, painful and more or less hard and there is a rather rapid diminution in the milk secretion. In *catarrhal mastitis* the *milk* becomes more or less altered and not much decreased, but the *udder* manifests few, if any, changes.

Because of these clinical differences the clinician, for convenience, recognizes *catarrhal mastitis* separately as the same process just indicated by that name, but under the collective head of *parenchymatous mastitis* he includes sero-hemorrhagic, croupous, purulent and croupo-purulent mastitis which show hot, painful swellings and rapid diminution in milk secretion. In addition he recognizes interstitial mastitis and gangrenous mastitis, which complete his classification, *i.e., catarrhal mastitis, parenchymatous mastitis, interstitial mastitis,* and *gangrenous mastitis.*

By use of the terms udder-infection, stroma-infection, and milk-infection (parenchyma-infection) in lieu of mastitis, interstitial mastitis, and parenchymatous mastitis respectively, terms with which we are more familiar in this country, the author keeps uppermost in the student's mind the fact that the actual presence of the bacteria in the various parts of the udder, and the primary lesions produced by their harmful influence, are the fundamental factors to be overcome. Not only are they to be overcome by the scientific application of prophylactic, medicinal and surgical remedies by the veterinarian, but mastered by elimination of the causative agents, the bacteria, and repair of the primary lesions induced by them, through the agency of that complex reparative process of the body called an inflamma-

tion, which is a reaction of the body to the action of an irritant, or an attempt on the part of the body to repair an injury.

Thus the inflammation or mastitis, by the various factors pertaining to it, *i.e.,* (1) alteration of tissue (primary lesion, degeneration, desquamation, necrosis), (2) emigration of leucocytes (fluid and cellular exudates, phagocytosis), or (3) proliferation of connective tissue (repair by healing, false regeneration), *is but the expression of the presence of the fundamentally interesting udder-wound, or udder-infection, be it stroma-infection or milk-infection.*

The foregoing co-ordination of terms used by Professor Wall with those most familiar to our literature precludes the possibility of failure on the part of the early student of pathology to grasp the complete significance and full appreciation of Professor Wall's treatise on the udder-inflammations of the cow.

By the use of [ ] I have endeavored to include such available data as tend toward a closer application to conditions prevailing in this country, and at this time.

WALTER J. CROCKER.

PHILADELPHIA, PA.,
January, 1918.

PREFACE

THIS work is based upon the investigation of milk of diseased udders and the subsequent autopsy of such diseased udders.

The material for these examinations consisted principally of samples of milk and specimens of udders sent to the High School for the investigation of tuberculosis.

In addition to this material, milk samples were obtained from acute udder-affections which were not tuberculous. Most of the material examined, however, was of a tuberculous character. The number of non-tuberculous udders amounted to ca. 50.

In order to study the kinds of udder-infections to be taken up not less than seventy bacterial isolations were made from milk and udders by the plate method. The culture medium was serum-gelatin-agar or serum-agar. Gelatin plates were poured in only a couple of cases and then for a special purpose.

For study of the histological changes in the diseased udders thirty different preparations were examined. Most of them were cut with the freezing microtome immediately after fixation in formalin. The remainder, ca. 10 cases, were cut after imbedding in paraffin. The sections were stained with hæmatoxylin and eosin or after the method of Van Gieson. In order to study the bacteria the sections were stained with carbolmethylene blue or after the method of Weigert or Ziehl.

Concerning the clinical description I am supported by clinical material from my veterinary practice (35 cases), and upon the cows with udder tuberculosis, purchased by the Veterinary High School, in which I produced mastitis by artificial infection. In addition other veterinarians have

contributed data on the clinical symptoms of the questionable or uncommon forms of mastitis.

To all colleagues who have assisted me in my work through material submitted or written information I extend my heartiest thanks. Finally I wish to express my deepest gratitude to my highly esteemed teacher, Professor J. Svensson, who has placed cows at my disposal and guided me in the technic of the bacteriological and pathologic-histological investigations.

SVEN WALL.

STOCKHOLM.

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MASTITIS OF THE COW

CHAPTER I

THE UDDER OF THE COW

Anatomical and Physiological Review

Anatomy.—The udder is a tubulo-alveolar gland. Like other glands, it consists of a true secreting part, the parenchyma, and a connective-tissue framework, called the stroma.

The parenchyma consists of extensively branched canals or passages as fine as a hair, called tubuli. At their ultimate extremities or blind end they show vesicular dilatations (tubulo-alveolar gland type) which gradually converge into larger collecting ducts that finally terminate in cavities in the teats as large as a hen's egg, called milk cisterns. The milk cisterns empty from the ends of the teats through a canal the size of a match, called the teat canal.

The walls of the finest tubules consist of a single layer of cubical epithelial cells. The collecting tubules, cisterns and teat canals are lined with several layers of epithelium.

The parenchyma is divided by the stroma into round lobules as large as a pea, within which the above mentioned different tubules are imbedded in a very thin, fine connective-tissue network called the intralobular connective tissue (Fig. 1). The part of the stroma which separates the single lobules is called the interlobular connective tissue. From this as a framework the fine intralobular connective tissue is given off. Blood-vessels and nerves lie in the stroma.

The intralobular connective tissue is made up of fine fibrillary connective-tissue tufts containing large lymph spaces. The interlobular connective tissue on the other hand is made up of firmer fibrilla showing only a few small spaces.

The collecting tubes are surrounded and joined by connective tissue which becomes increasingly firmer and thicker and which constitutes an effectual firm connective-tissue membrane around the epithelium of the cisterns and the teat canals.

The gross anatomical structure of the udder of the cow consists of four parts, the two lateral halves which are separated from each other by loose connective tissue, a left and a right half. These halves are again divided into two parts, an anterior and a posterior quarter. These are very firmly bound together by connective tissue.

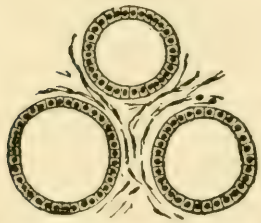

Fig. 1.—Cross section of three tubuli (× 150).

The udder receives blood through the external pudic artery which is a branch of the crural artery. These external pudic arteries divide into two branches, one for the anterior and one for the posterior quarters. The blood is returned through two blood-vessels: (1) the milk vein or subcutaneous abdominal vein, which runs anteriorly to the internal thoracic vein, and (2) the external pudic vein, which passes posteriorly (Fig. 2).

The lymph-vessels of the udder are numerous and pass posteriorly to the supramammary lymph-glands and from here out through the internal lumbar-glands to the thoracic duct.

Physiology.—The udder secretes the milk. It functionates periodically and the period depends upon pregnancy; thus milk secretion begins with great quantities with parturition and continues for ca. 7–8 months. After this

time the milk decreases, the cow loses tone and finally with difficulty gives a negligible quantity of milk which is grayish-yellow, thin and watery.

In general the first milk secretion is produced through reflex stimulations at parturition. The normal milk secretion is only produced through birth.

Milk secretion can be produced however under other circumstances, as during œstrum. This is of little importance, as a rule, and the udder soon loses its tone again (spontaneous milk secretion).

Experimentally it is possible to produce an atypical milk secretion through stimulation of the corresponding nerves.

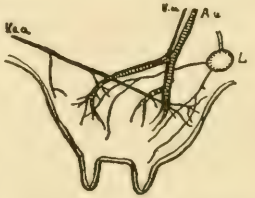

Fig. 2.—Vessels of the udder (after Moussu).
V.e.a. = Anterior epigastric vein.
V.u. = Vena uberi.
a.u. = Artery uberi.
l. = Supramammary lymph-gland.

The above mentioned method of producing milk secretion is maintained through mechanical stimulation by the sucking of the calf or by the hand of the milker. Without these mechanical factors the milk quickly diminishes.

The milk secretion is therefore introduced by parturition and maintained through mechanical stimulation (milking).

This mechanical stimulation however cannot sustain the milk secretion indefinitely, as the milk secretion diminishes usually about two months before the next birth.

The lactation period can only be extended by mechanical stimulation when no impregnation has taken place. This can be induced for from fifteen to eighteen months, especially after castration; in every case however a gradual diminution occurs.

In this condition the blood supply is decreased and the udder is poor in liquid and dry (diminished lymph).

The reflexly produced activity of the udder cells disappears simultaneously with the blood supply and in con-

sequence of this lessened nervous stimulation of the gland cells and decreased nutrition a condition of diminished milk secretion results.

The quantity of milk is greatest during the first month after parturition but gradually decreases afterward. The udder decreases in circumference as it is no longer distended with milk and becomes small and quiescent or in the dry or alactiferous state.

In consequence of the diminished work and nutrition the organ undergoes a process called atrophy in pathology but which is physiological in this case. The cell elements become smaller and their number decreases, since part of the cells become retrogressively altered, fatty degenerated and desquamated. The tubules and consequently the lobules become smaller in this way. At the same time, as an indication of retrogressive alterations, great numbers of leucocytes emigrate to the intralobular connective tissue where they assist in the work of absorption.

The atrophic gland cells give off other products of secretion. This secretion is no longer normal milk, it is grayish-yellow and thin, not infrequently viscid and contains a large quantity of albumin so that it coagulates on boiling. Moreover it is rich in cells, epithelial cells and especially in emigrating leucocytes which are often laden with fat droplets. The secretion is resorbed for the most part, in which work the leucocytes are directly responsible as they re-enter the blood-vessels laden with fat droplets. The atrophic gland cells are capable of producing a yellow coloring matter within their structures which lends a distinct dark yellow color to the cut surface of the yellow udder (yellow atrophy).

At the next parturition the condition is immediately changed. The cells become stimulated to secretion and the atrophic udder is again well nourished with blood, consequently the cells very quickly become functionable again and the milk, especially, very soon becomes normal. The

atrophic pigmentation and likewise the leucocytic infiltration disappear.

The milk of the first few days, the so-called colostrum, has an albumin and leucocytic content of great similarity to the milk of the atrophic udders, the "gelt" milk.

During this quick change from the dry to the lactation state circulatory disturbances may easily occur. The functional hyperæmia can frequently be so increased that an abnormal transudation occurs inducing œdema of the udder. Following this condition small capillary hemorrhages may occur in the intralobular connective-tissue spaces from which blood may empty into the milk-ducts, mix with the milk and cause bloody milk.

Following these changes functional disturbances may also develop. It is very probable that the atrophic cells also secrete other abnormal products, such as virulent toxins although in insignificant quantities. Following parturition however large quantities of the same toxin may be produced due to the altered condition which may lead to a general intoxication, milk fever. This disease is treated by air inflation with localized cell paralysis due to the effect of pressure.

Summary

I. Anatomy of the udder:
   - A. Microscopic structure.
     1. The parenchyma of the udder.
     2. The stroma of the udder.
   - B. Gross anatomical structure.
     - Arteries and veins.
     - Lymph-vessels.

II. Physiology of the udder:
   - A. Milking period.
   - B. Incitation and maintenance of milk secretion.
   - C. Diminution, dry or alactiferous udder, "gelt" milk.
   - D. The udder at the time of parturition.
     - Circulatory disturbances.
     - Functional disturbances.

CHAPTER II

MASTITIS IN GENERAL

DEFINITION.—Inflammation of the udder or mastitis is a local disease of the udder of a reactive character which manifests itself by an active hyperæmia, emigration of cells, and proliferation of connective tissue.

ETIOLOGY.—Mastitis is brought about through harmful local influences. These harmful influences are of two kinds: (1) physical, mechanical nature; external force; (2) chemical nature, bacterial toxin; infection.

PATHOGENESIS.—The part of the udder exposed to these harmful factors can consequently undergo retrogressive alterations and indeed become so injured that a more or less progressive death of cells supervenes as the primary lesion.[1] The adjacent living tissue subsequently reacts with an inflammation. In this condition the emigrating cells, especially the leucocytes and connective-tissue cells, rid the local diseased area of dead cells and foreign bodies that might be present. The connective-tissue proliferation which occurs at this time is a reparative process and gradually fills all defects existing in the tissue by the process of healing.

This process is supported by a rich blood supply to the diseased part called hyperæmia, following which the physiological transudation is increased, and this increased transudate, together with emigrated cells, possibly present foreign bodies and dead cells, is called an exudate.

The real position of the inflammation is in the stroma. The parenchyma is secondarily affected in mastitis following alterations in the stroma.

In consequence of the difference in the structures of the

[1] Like Ribbert, I do not classify the retrogressive process with inflammation, but I maintain that it is the incentive factor of the inflammation, which therefore may best be called the primary lesion. [Alteration.]

stroma, the intralobular connective tissue is the most severely affected, as it is the most loosely constructed and richest in juices. These spaces become filled with exudate which consists principally of leucocytes and connective-tissue cells. In this condition the tubules become pressed together and the epithelial cells undergo desquamation. Part of the exudate is forced into the tubules and mixed with the milk, which consequently becomes altered in its composition, especially in its cell content. At the same time a proliferation of the connective tissue takes place with thickening of the intralobular connective tissue. Similar alterations but of a lower grade take place in the remaining stroma of the inflamed part.

During the lactation period the udder is most rich in juice and most sensitive, and the inflammations are therefore more severe and often malignant. The dry or alactiferous udder, which is comparatively poor in fluid, is however less sensitive toward the external influences, and its inflammations are therefore milder and more benign.

Termination.—When the irritating matter has been removed by the leucocytes, inflammation recedes. The exudate is resorbed, leucocytes re-enter the blood- and lymph-vessels, and cell infiltration disappears or is reduced to a minimum. The intralobular connective tissue becomes thinner and the tubules are again dilated and enlarged.

The original circumference of the tubules is not attained however, as the intralobular tissue has become thickened by proliferation of the connective tissue, which does not completely reduce following a severe inflammation. As a result of inflammation there is usually a connective tissue proliferation of varying degree which may terminate as sclerosis with subsequent diminution of tubular circumference or atrophy.

Classification.—The causes of mastitis may be divided into: (1) mastitis caused by external force; (2) mastitis caused by infection, infectious mastitis.

CHAPTER III

MASTITIS CAUSED BY EXTERNAL FORCE

This mastitis may be divided into injuries and wounds.

INJURIES OF THE UDDER

An injury of the udder is a destruction of the udder tissues through a more or less uninjured skin covering.

Causes.—Injuries of the udder may be produced by a blow, by squeezing or by being trod upon.

One must discriminate between the primary effect, that is the primary lesion produced by the direct effect of the external force, and the subsequent inflammation.

Primary Lesion.—The force can produce an immediate necrosis of the softer cells, especially the epithelial cells, in a circumscribed area and rupture of blood- and lymph-vessels with an extravasation of blood and lymph into the intralobular connective tissue particularly. This hemorrhage can also get into the milk passages and give rise to bloody milk. As a rule the hemorrhage soon stops. As the tissue becomes distended with blood and lymph the pressure becomes so great that further hemorrhage is mechanically lessened or prevented. Furthermore the destroyed cells liberate fibrin ferment which aids in coagulation of the blood, thus preventing further hemorrhage. Should the force strike an exposed part which already shows a blood infiltration, a progressive necrosis of the rather poorly nourished cells can easily take place. Seldom is the udder tissue alone affected, as usually the skin is involved at the same time and manifests superficial abrasions of the epidermis and swelling and blood infiltration of the corium and subcutaneous connective tissue.

These alterations in the udder tissue then call forth an inflammation with emigration of leucocytes. This in-

flammation clears the tissue of extravasated blood and necrotic particles and fills the defects made in the tissue by necrosis and its resorption, with connective tissue.

Similar phenomena occur in the skin as dermatitis when it becomes involved.

Termination.—After resorption of the exudate in the inflamed area, more or less sclerosis remains followed by atrophy of the tubules. If the skin has also been involved in the inflammation, it is no longer movable but is firmly adhered to the udder.

If there is a wound in the skin it may easily become infected. This infection can possibly reach the injured area in the udder through the lymph-vessels and in that way give rise to an infectious mastitis.

Clinical Symptoms.—We see immediately or soon after the impact of the force, as a rule, the formation of a moderate circumscribed swelling which is the primary lesion. In the course of one day the swelling increases, becomes hot (hyperæmia), painful and somewhat firm following blood coagulation and cellular infiltration.

After several days the swelling begins to recede and gradually diminishes, after which, however, a somewhat firmer, more circumscribed nodular formed area remains, due to connective-tissue induration.

Treatment.—The condition is not serious. Usually no treatment is necessary. The process heals out rapidly.

In the first few days one can reduce the hyperæmia and hemorrhage by the use of cold-water treatment (vasoconstriction). One can apply a cold water compress to the udder by a suspensory bandage placed around the middle. If the skin is injured one can prevent infection of it by the use of a Burrow's solution compress.

When the swelling begins to recede one can assist the treatment by the use of a skin irritant, as concentrated iodine solution rubbed in as a stimulation to resorption.

WOUNDS OF THE UDDER

ETIOLOGY.—Wounds of the udder may be produced by blows, or stabs as by a pitch-fork, hooking, or from a surgical operation such as an incision in an abscess.

This brings us to the discrimination between the primary lesion and the subsequent inflammation.

The wound ruptures the vessels and milk passages, following which the walls of the wound become infiltrated with blood, lymph and milk which flow outward through the mouth of the wound. The hemorrhage may be more or less copious. It is greatest following incisions and least in contused wounds, that is in wounds produced by dull objects, as a thrust from a horn. In contused wounds the vessels of the neighborhood are so injured that thrombosis occurs in most of them. This prevents hemorrhage but predisposes to necrosis.

INFLAMMATION.—The reactive inflammation begins in the immediate vicinity of the wound, that is, the walls of the wound, where a wall of emigrating cells [phagocytes] is formed which clears the wound of dead tissue and foreign particles. Around this a connective-tissue wall is formed which closes the ruptured tubules and from which connective-tissue cells rapidly proliferate until the wound aperture is gradually filled with young succulent loose connective tissue, granulation tissue.

TERMINATION.—In the most favorable cases the wound heals without any complication. The newly formed connective tissue fills the wound aperture, and the surface is finally covered by regeneration of the epidermis from the margin of the wound. The exudate is resorbed and the loose connective tissue strongly contracts, finally leaving a white fibrous line or scar.

As a rule, however, udder wounds become complicated with infection. In these conditions healing is usually delayed, necrosis of the connective tissue is more widespread

and not infrequently such a condition can develop into a more protracted exudative gangrene.

TREATMENT.—Our treatment is to avoid an infection when possible and to check its progress when one has taken place.

The wounds are thoroughly disinfected. In the beginning a Burrow's solution compress is applied to the udder (suspensory bandage). Infected wounds are freely opened so that the exudate may have free drainage, necrotic parts are removed, following which a scrupulous antiseptic treatment is carried out.

CHAPTER IV

INFECTIOUS MASTITIS IN GENERAL

Ports of Infection

The causes of infection may enter the udder: (*a*) through openings in the udder, and (*b*) through the blood-stream.

The openings which may serve as ports of infection to the udder are of two different types: (1) natural: the teat canals, and (2) morbid: wounds.

THE TEAT CANAL AS A PORT OF INFECTION

Infectious material enters through the teat canal, so that first the milk and indirectly through it the tissue become infected.

The milk is a nutritious fluid and contains water, salts, albumin, carbohydrates, and fats. It is an excellent source of nourishment for bacteria and therefore difficult to preserve, especially in summer, since it easily undergoes acid fermentation in vessels.

The udder keeps the milk warm, like a thermostat, and offers very favorable conditions for bacterial development.

Under normal conditions, however, the milk in the udder is practically sterile. The reasons for this are of two types: (*a*) physical nature, and (*b*) biological nature.

Physiological causes of sterility of milk in the udder. The teat canal is closed between milkings by bundles of smooth muscle tissue imbedded in the walls which maintain a definite moderate tension. Thus under physiological conditions the external opening of the teat canal is small, usually rendering infection more difficult.

Furthermore, the teat canal is directed downward, or to the side when the cow is lying, but never upward, as the

dorsal position is not physiological to the cow. Thus usually the bacterial infection takes place in a direction opposite to that of gravitation, never in the direction with it, which usually is an obstacle to infection.

Finally the milk stream passes only in one direction, outward from the udder through the teat canal by sucking or milking. A retrogressive stream does not occur. The udder is mechanically distended by the milk and collapses like an empty glove after milking, without the influence of any in-sucking force. Thus the infection must enter against the stream, never with it.

Biological causes for sterility of milk in the udder. In spite of the before mentioned obstacles to infection one or more bacteria may gain entrance, but these may be taken up and destroyed by the ever-present leucocytes which are more or less numerous. The leucocytes play a very important part inasmuch as they inhibit the invasion of the milk by bacteria. [The normal healthy udder of the cow harbors bacteria throughout its whole extent (Ward). The healthy udder always gives milk which is entirely bacteria free (Kitt., vol. i, 2d ed., p. 227).]

Predisposing factors to infection through the teat canal. These factors are of two types: (*a*) physical nature, and (*b*) biological nature.

Predisposing Causes of a Physical Nature.—Arrest of milk secretion is produced by neglecting to milk or from poorly regulated milking hours. For the same reasons the cisterns can become so distended with milk that the pressure finally overcomes the tension of the sphincter muscle and the teat canal fills with milk which continuously drops from the end of the teat. A rapidly growing or motile bacillus can now easily gain entrance against the stream, grow in the milk in the teat canal, and finally infect the entire milk.

A paralysis of the sphincter muscle following age, as

in old milch cows, or a more severe affection, as a chill or fever,[1] causes the same alteration, and dilatation of the milk canal takes place and predisposes to infection.

Infection may be induced by the introduction of instruments, as a milk tube in the teat canal, or spontaneously with foreign bodies, as a straw which may be forced into the teat canal when the cow lies down.

Predisposing Factors of a Biological Nature.—When a very virulent rapidly growing bacillus gains access to the mouth of the teat canal it can always find spaces in that passage containing moisture from the physiological capillaries in which it can grow. Through its own virulence it can withstand the leucocytes (negative chemotaxis) [aggressins], and infect the milk. This explains the distinctly contagious character of many cases of mastitis.

Prophylactic Measures Against Infection Through the Teat Canal.—Avoid arrest of the milk secretion by well-regulated milking hours and punctuality in milking. Use the milk tube cautiously. Keep the teats clean and dry. In cases of contagious mastitis isolate the diseased individual, milk it last and disinfect the morbid milk in order to avoid the possibility of spreading the infectious material in the cow stable.

UDDER WOUNDS AS PORTS OF ENTRANCE

Udder wounds may already be infected from the beginning, that is, the infectious material is injected at the same time that the wound is made. Such is often the case in stab wounds. In other cases a wound may be sterile at first and become infected later.

In fresh wounds the exterior of the wound itself and also the milk through the ruptured tubules may become

[1] Both appear to be followed by paresis of the involuntary muscles which among other things produces diminished peristaltic action.

infected. In old wounds this is prevented by the inflammatory connective-tissue formation.

Protection of Wounds Against Infection.—The leucocytic wall forms the most important protection against infection of the wound. But since the wall of leucocytes is only an evidence of inflammation, that process plays a very important part against infection.

Finally the inflammatory connective-tissue wall forms a very good obstacle to the progress of infection, as it circumscribes it.

Predisposing Factors to the Infection.—The nutrient fluids, blood, lymph, and milk which infiltrate the udder wound, predispose it to infection more than any other wound because of the favorable nutrient conditions for the infectious organisms and because the wound itself is a good thermostat.

The greater the blood infiltration, the more widespread the death of cells, and the more saturated with fluids the wound is, just so much better it is for infection. It is broken down by the bacteria which as a rule live more easily on dead than on living media. In the latter case they are more directly exposed to healthy phagocytes.

THE BLOOD AS A PORT OF INFECTION

In order that the blood coming into the udder may be a port of infection, it is necessary that we have a blood-infection, that is, that the infectious organisms be in the blood stream.

Blood-infection.—The blood can become entirely or partially infected, that is, general blood-infection (bacteræmia), or partial blood-infection.

A partial blood-infection is possible in this way, that the infectious organism be deposited in the organs or be transported to the organs from the blood by leucocytes.

The local blood-infection, which is of importance to the udder, is through infection of the blood of the aorta.

Causes.—A blood-infection can be produced in different ways. Infectious material may be picked up by the leucocytes in an infected area in the intestines or in other organs rich in bacteria and be carried by them into the blood stream. In a wound infectious organisms can grow directly in ruptured vessels. In the infection of an organ the primary lesion (necrosis) may become permeated with infectious organisms, which surround the vessels of the primary lesion, inducing deep necrosis, so that a blood-infection may take place through the walls of these vessels. Blood-infection can be induced through infectious ulceration of a vessel wall. Should the lymph become infected (lymph-infection) this fluid can finally infect the blood.

The usual causes of infection of the blood of the aorta is a lung-infection or an infectious endocarditis.

If the blood of other organs, for instance of a wound or of a uterus, as in infectious metritis, be infected the blood of the aorta can still remain sterile, since the infectious material is filtered out in the lungs. This is the rule for infectious organisms of positive chemotaxic action, while on the contrary infectious organisms of negative chemotaxic action easily pass through the lungs.

With infectious pneumonia usually a blood-infection is brought about through ulceration of a vein wall with emptying of infectious fluid material into the blood of the veins.

The exudate discharges itself into the vein gradually and thus divides itself minutely. It becomes further comminuted in its passage in the vessel by impact against the vessel wall, and through the action of the current eddies in the vessel ramifications. A drop of exudate discharged into the vein can become so finely divided in this way that it can be distributed over an incredibly large area.

Infection of the blood stream is not of long duration as the blood soon deposits the infectious organisms and thus purifies itself again. Infection of the blood can recur, however.

An acute fatal general blood-infection like anthrax naturally cannot give rise to a local diseased process, such as inflammation of the udder. A non-fatal generalized blood-infection can, however, produce different infectious local disease processes, such as pneumonia, pleurisy, arthritis, nephritis, and perhaps mastitis.

Infection of the Blood of the Udder.—The possibility of one of these emboli reaching the udder is very great, since both external pudic arteries are fairly large, have no important curves, and are incipient at the aorta. [External iliac, femoral artery, external pudic or mammary artery.]

As the aorta runs on the left side [anterior part], the most direct course is to the left half of the udder which is therefore most exposed to a blood-infection. It is clear, however, that the situation and the time during which the emboli are passing in the blood are of great importance. If, for instance, a cow stands with her posterior extremity inclined toward the left, the right external pudic artery is the most direct course. For the emboli to reach the posterior quarters the most direct way seems to be through the external pudic arteries.

Thus the left quarters of the udder are more exposed to a blood-infection than the right, and the posterior quarters more so than the anterior. The left posterior quarter therefore is the most severely exposed.

The small infectious emboli, if they be sufficiently large, may be caught in the capillaries or fine arterioles and at these points produce a thrombus with infectious necrosis and the formation of infectious metastatic foci. In other cases very small emboli may be taken up and carried into the loose intralobular connective tissue where the leucocytes

degenerate, remain and die when the infectious organisms are the stronger. Here a metastatic infectious focus is produced. On the other hand, if the leucocytes are the stronger, the infectious organisms are destroyed without the production of tissue alterations.

In this manner, as a rule, through infection by the blood to the udder a great number of small foci, distributed throughout the connective-tissue stroma of the udder, are formed.

Infection of the Udder in General

The infection of the udder may be divided[2] into *milk-infection* and *stroma-infection,* depending upon the position of the infectious organisms.

milk-infection [*Parenchymatous Mastitis*].

Definition.—*Milk-* or *parenchyma-infection* is an infection of the udder in which the bacteria are only found in the milk, or rather in the parenchymatous passages.

Since the parenchymatous tubular system of one quarter does not connect with that of the other quarter the condition remains confined to the quarter infected.

Ports of Infection.—The milk-infection [parenchymatous mastitis] may be primary or secondary.

The primary milk-infection is produced by infection through the teat canal or through a ruptured tubule in a fresh wound.

The secondary milk-infection is a subsequence of stroma-infection [interstitial mastitis] with extension of an infectious focus in the stroma to a milk passage, ulceration through the wall of the tubule and into the milk tubule.

Milk-infection causes a local condition in the udder as well as general conditions.

[2] I have made this division, since it appears to me better than the old divisions indicated by the position of the disease.

THE LOCAL CONDITION.—We must here distinguish between the primary lesion and the subsequent inflammation.

PRIMARY LESION.—We distinguish between the quantity or extent, and the quality or character of the primary lesion.

QUANTITY.—The primary lesion may be more or less extensive. It may be circumscribed upon a greater or smaller part of a quarter of the udder or it may be diffusely spread over the entire quarter.

Naturally it always begins by affecting a localized part but can become diffuse through spreading of the infectious organisms in a comparatively short time (a few hours, days or weeks).

The factors which may circumscribe the infection are mechanical or biological in character.

The mechanical inhibiting influences are the milk stream and gravity. The infectious organisms must develop against the current of the milk secretion, which is usually a fairly effective obstacle to an extension of the infection. Greater difficulty is experienced by the infectious organisms in developing in the milk-ducts in the opposite direction to gravity. For this reason, therefore, they grow at first in the horizontal milk passages on either side of the teat bases, especially in the part behind the teats of the posterior quarters and before the teats of the anterior quarters. The progress upward is more difficult and more protracted.

The inhibiting biological influences against the bacteria are the leucocytes.

If the organisms grow slowly phagocytosis usually prevents spreading of the infection.

The primary lesion more or less quickly produces an inflammation in the region, with emigration of leucocytes which may mechanically prevent further infection. It creates a cell stopper against the infection, as it were.

These factors adequately support each other, that is, a localized primary lesion is caused by an organism of low virulence and slow growth, while, on the other hand, the diffuse primary lesion is caused by an organism of high virulence and rapid growth. When the virulence is extremely high inflammation may be retarded or prevented.

Quality.—The alterations in the primary lesion are of a retrogressive type, and are usually of degenerative or necrotic character. These retrogressive changes, however, may vary in degree. In the mildest cases they may consist only of degeneration of the epithelium.

Fig. 3. — Milk-infection [parenchymatous mastitis]. Staphylomycosis. Severe primary lesion, with diffuse gangrene (150). Nuclei indistinctly or not at all stained due to necrosis. The intralobular connective tissue is distended with red blood cells following hemorrhage. The tubules are compressed and contain desquamated epithelial cells. Numerous staphylococci were demonstrated in the tubules by using bacterial stains.

The epithelium may however become necrotic and a croupous membrane form. If the connective tissue becomes necrotic a deep diphtheritic membrane is produced. Greater portions of the udder may become necrotic, as the single tubules, or groups of these tubules, forming nodular necrosis. The entire part of the udder affected may rapidly become necrotic or develop into diffuse gangrene (Fig. 3).

The quantity and quality of the primary lesion depend upon the character of the infectious organism and the condition of the udder. The more virulent the organism, the greater is the area and the more severe the character of the primary lesion.

Organisms of the same virulence however produce a greater primary lesion after parturition than during the gestation period. Under identical conditions, that is, the same virulence of organisms and same time after parturition, the primary lesion is greater in good milch cows than in those that are poor milkers. These circumstances are

dependent upon the quantity of milk, which is great in one case and small in the other, and thus provides a more or less favorable condition for existence for the bacteria.

The milk is also changed by the primary lesion. The more widespread and severe the alteration in the udder the greater are the changes in the milk. The casein and fat content of the milk is lessened by degeneration of the parenchymatous cells. The milk is rich in albumin in consequence of transudation from the blood stream, shows an increase in cells following desquamation of necrotic epithelium and is of a red color due to retrogressive metamorphosis of the red blood corpuscles.

The inflammation occurs as a reaction to the primary lesion.

Since the infectious organisms lie in the tubuli, quantities of leucocytes wander into the lumen of those structures. The milk is therefore rich in cells (Fig. 4).

Fig. 4.—Milk-infection [parenchymatous mastitis] Colibacillosis. Acute inflammation (× 150). The epithelial cells are swollen and show weak nuclear staining (degeneration). At a few places there are a few layers of cells. Intense cell infiltration in the intralobular connective tissue and severe emigration of cells into the tubules. A few colon bacilli were seen in the tubules by using bacterial stains.

Quantity.—If the primary lesion is circumscribed, the inflammation is circumscribed, and if the primary lesion is diffuse, the inflammation is likewise diffuse. (Except in diffuse necrosis. See later.)

Quality.—The exudate is of a suppurative purulent character. The inflammation, following the character of the primary lesion, is purulent catarrhal, purulent croupous, or diphtheritic, sequestrating or apostematous.

The more virulent the infectious organism the longer the inception of the inflammation is deferred, and thus the greater the primary lesion will be. The strongly virulent infectious organism works repellently upon the leucocytes (negative chemotaxis).

The necrosis produced by the infectious organism how-

ever mechanically hinders this repulsion, and the necrosis also prevents a strong attraction for the leucocytes. In defence of the body the leucocytes are finally attracted to the necrosis, and now the infectious organisms are subjected to the action of these cells which inhibit their growth and toxin production. The weakened infectious organisms can then be rendered innocuous or killed or be dislodged with the necrotic masses and be discharged with the milk.

Through desquamation of a necrotic, croupous or diphtheritic membrane a milk-infection can develop into a secondary stroma-infection. Following such desquamation a wound is formed which can become infected (wound-infection.) In this manner a sequestrating purulent inflammation with nodular necrosis is produced, constituting a secondary stroma-infection or interstitial mastitis. Following complete softening of the sequesters the area is changed into an abscess with a thick fibrous wall, which is produced by inflammatory proliferation of the adjacent connective tissue.

In diffuse necrosis mastitis as a sequestrating inflammation naturally cannot proceed further than the periphery of the diseased quarter of the udder.

Milk.—The milk, which is changed by the primary lesion, is further altered by the inflammation. It is extremely rich in cells and therefore purulent, thick and pulpy. Its color is yellowish-gray, due to the presence of cells, and it often contains croupous membranes as thick yellow shreds. The reaction is somewhat strongly alkaline. The milk never contains clumps of casein. [Kitt and Jensen describe casein clumps in morbid milk.]

Complication with Saprophytic Invasion.—Under the usual conditions pathogenic bacteria, but not saprophytes, may invade the milk in the udder. If the latter do gain entrance they are taken up by the leucocytes and destroyed. It may be different in other conditions such as

an udder-infection. The leucocytes which are dangerous to the saprophytes may be called back by the pathogenic organisms, and then the saprophytes may develop unhindered.

They cannot attack living tissue, but the necrosis produced in the primary lesion constitutes an excellent culture medium for them, and consequently undergoes putrefaction. We may call this process exudative gangrene.[3]

These saprophytes are large, anaërobic, rod-shaped putrefactive bacteria.

A saprophytic invasion of this type may easily occur in croupous mastitis, since a shred of fibrin can remain hanging in a teat canal. A shred of this kind can then become permeated by saprophytes which grow in and through it, so that the shred may constitute a good bridge for the infection.

Following such a saprophytic invasion with exudative gangrene, the milk becomes stinking, homogeneous and thinly fluid, since the necrotic cells and the fibrin shreds dissolve during putrefaction, and the leucocytes are killed by the poisons of putrefaction.

GENERAL CONDITIONS

Blood-infection.—The primary lesion may be permeated with the growing pathogenic organisms, which induce deep necrosis with destruction of the blood-vessel walls, so that the bacteria may enter the vessel and produce a generalized blood-infection or septicæmia. This secondary blood-infection, if not overcome by the defensive elements of the blood, can induce secondary lesions, as an infectious pneumonia or arthritis.

Death caused by an acute general blood-infection is due to septic intoxication. The external symptoms of a

[3] These necrotic parts will be thrown off with the croupous or diphtheritic exudate and constitute a part of that exudate.

general blood-infection or bacteræmia are the same as in the septic intoxication.

Intoxication.—From the diseased quarter of the udder a stream called the exudative stream passes into the milk, and another stream called the resorption stream passes into the blood.

This resorption stream carries a quantity of toxin produced by the infectious organisms, which induces the more or less distinct appearance of intoxication, which we call septic intoxication because it is caused by infectious material or the toxins of pathogenic bacteria.

Septic intoxication presents a collection of general or nervous symptoms which we call fever symptoms [complex symptom of fever]. They are:

1. The heart shows an increase in the frequency but a decrease in the force of the beat.

2. Respiration shows an increase in frequency but a decrease in volume (dyspnœa).

3. The skin is warm, due to hyperæmia following vasodilation.

4. The voluntary muscle shows diminution of power [trembling, decubitus].

5. The smooth muscle likewise shows a diminution of power. Peristalsis is especially weakened or ceases entirely, inducing indigestion or impaction. [It may alternate with increased activity and diarrhœa.]

6. The secretion of sweat is more or less increased.

7. The body temperature is increased. [It may be normal or decreased.]

8. Consciousness is diminished.

Fever begins however with a stage showing the opposite symptoms, which resemble the reflexes produced through stimulation due to irritation by cold. The clonic-tonic spasms of the voluntary muscles called shivering, are very prominent, likewise the coldness of the skin due

to anæmia of the skin following vasoconstriction [chill]. In this stage the body temperature ascends as a result of increased heat production by muscle spasms and the diminished heat radiation due to anæmia of the skin. [Visceral congestion, retention of waste products, and increased retrogressive cellular metabolism also play a part in heat production.]

In consequence of the high temperature and the great heat loss through hyperæmia of the skin, combustion and consumption of energy are very great in fever.

Death may be produced in septic intoxication by paralysis of the heart or the vital nerve centres by the effect of toxins.

In exudative gangrene a putrid toxin or putrefactive poison [cadaverin] is formed which produces putrid intoxication when resorbed. An intoxication of this kind is called septic-putrid intoxication [septico-sapremia].

Putrid intoxication also presents symptoms of fever. Death may easily occur in this condition, and it is produced with distinct paralysis of the nerve centres, indicated by collapse, with subnormal temperature, muscular paralysis, and loss of consciousness (narcosis).

Termination.—Milk-infection or parenchymatous mastitis is seldom fatal. The cause of death is septic or septic-putrid intoxication.

This general intoxication can, however, usually reduce the milk secretion and nutritive condition of the animal.

The udder can heal after desquamation and removal of the necrotic parts. Healing takes place with loss of substance which is principally replaced by connective tissue with atrophy of the milk-producing parenchyma.

The duration of the condition depends upon the quantity and quality of the primary lesion and may be from four days to one month or even longer, as is the case in nodular necrosis.

Not infrequently, however, the infection is progressive in spite of the inflammation. The infection spreads further to the healthy lobuli and induces a new primary lesion with its subsequent inflammatory reaction.

The course therefore is more protracted. The condition may last from a month to a year and the terminal result is a diffuse sclerosis and atrophy of the parenchyma.

The functional capacity of the udder becomes more or less reduced following the inflammatory process induced by the milk-infection. This deficiency may be overcome, however, through vicarious hypertrophy of the unaltered parts of the udder.

STROMA-INFECTION [*Interstitial Mastitis*]

DEFINITION.—Stroma-infection or interstitial mastitis is that infection of the udder in which the infectious organisms are only found in the stroma or interstitial connective tissue.

The pathogenic organisms invade the lymph spaces of the intralobular connective tissue.

Since the connective-tissue system binds the different quarters of the udder together, the stroma-infection can spread from one place to the neighboring tissue, while the milk-infection, on the other hand, is confined to the infected quarter.

The stroma-infection may be divided into two principal groups:

1. Stroma-infection of an acute character.
2. Stroma-infection of a chronic character.

STROMA-INFECTION OF AN ACUTE CHARACTER [*Acute Interstitial Mastitis*].—These cases depend upon quickly acting infectious organisms, which in their active way may produce an acute, never typically chronic, process.

The point of infection is one or more wounds. The blood is seldom the source of infection and this infection

in the udder may be primary in the stroma, although not coming from the milk into the stroma. The wound is caused by an external violence and is therefore an external wound which opens through the skin. The pathogenic organisms come into the wound from the exterior. The infection through the blood, due to its position in the udder, is always primary in the stroma.

Furthermore, this infection can be secondary in the stroma when it comes into the stroma from the milk. The wound, or the wounds, are caused by milk-infection with development of necrosis after the occurrence of desquamation or secondary wounds. These internal wounds are infected by the same infectious organisms which cause the milk-infection [parenchymatous mastitis].

The location of the bacteria in these stroma-infections is in the wound secretions and in the primary lesion.

Local Condition.—The primary lesion extends over the surface of the wound and forms a superficial or more or less deep necrosis. The wound secretion is usually altered by the primary lesion. Following the necrosis of the vessels the lymph or plasma extravasation increases, the blood corpuscles remain for the most part in the vessels. The secretion is colored red by hæmoglobin due to necrosis of red corpuscles.

In stroma-infection, through the blood, more frequently larger or smaller embolic areas of necrosis are found.

The inflammation induces a severe leucocytic emigration into the connective tissue of the walls of the wound and into the wound. The wound secretion is suppurative. It contains a great quantity of cells, leucocytes and young connective cells, has a gray or yellowish-gray color and is somewhat thick.

Around the necrotic parts a dissecting cell-ring of leucocytes forms, which loosens the necrosis.

The loosened necrotic material can slough directly to

the exterior in external wounds, or in internal wounds be forced or desquamated toward the interior into the milk.

In nodular necrosis following milk-infection or in embolic necrosis following infection through the blood, the necrotic material cannot be gotten rid of as such, but it can be liquefied through the action of the infectious organisms and the cells of the inflammation (so-called suppurative softening). The pus formed in this manner can empty itself through the milk passages (in which the primary embolic stroma-infection thus induces a secondary milk-infection) or directly to the exterior through ulceration of the skin. The latter, ulceration of the skin, occurs however only in superficial nodules. In other cases complete or only partially liquefied areas of necrosis can become encapsulated following a stronger proliferation of peripheral connective tissue. This condition is then chronic.

The Milk.—In externally infected wounds which have not progressed to a milk-infection, the milk is only slightly or not at all altered. The quantity, however, is more or less diminished.

In internally infected wounds the milk possesses the same character as in the milk-infection [parenchymatous mastitis].

A complication with saprophytic invasion can occur and develop into an exudative gangrene. The exudate is stinking and less densely fluid.

Blood-infection.—*Bacteræmia.*—In addition to entering the milk, infectious organisms of a high virulence can enter the blood stream through the opened blood-vessels of a fresh wound or through the walls of the vessels injured in the primary lesion. In this manner an acute blood-infection can take place, which, if not fatal, leads to subsequent disease processes as infectious pneumonia or arthritis.

Intoxication.—Stroma-infection can also result in

septic intoxication, or develop into exudative gangrene and then septic-putrid intoxication with the same symptoms as before described, and these conditions may terminate in death.

Termination.—Stroma-infection or interstitial mastitis very seldom terminates in death. Healing is the rule. The wounds heal after sloughing of the necrotic parts, with more or less sclerosis and parenchymatous atrophy.

Nodular necrosis following milk-infection or the less frequent embolic necrosis may form chronic wounds which may induce a sort of relative healing through encapsulation.

Stroma-infection of Chronic Character [*Chronic Interstitial Mastitis*].—The action of the bacteria is of a chronic progressive character.

Ports of Infection.—These infections may take place through the teat canal, through wounds or through the blood stream.

In the latter case the infection is always primary in the stroma due to its position in the udder. Whatever the port of entrance may be the organisms are taken up by the leucocytes and carried into the lymph spaces of the stroma. Here the leucocytes soon become paralyzed and are necrotized by the bacteria.

Local Conditions.—The primary lesion consists of many single spreading areas of necrosis in the stroma around the point of infection in the teat canal, wound or at the point of infection from the blood stream.

Around each primary lesion the inflammation produces a strong ring of leucocytes and outside of this a wall of new-formed connective tissue.

Thus the inflammation produces many small areas or nodules the size of a pin's head or smaller (Fig. 5).

The progressive character, that is, the chronic enlargement of the primary lesion, the necrosis, is diagnostic of this infection. The primary lesion is chronic, as it were,

and usually is not circumscribed by an inflammation. The necrosis develops slowly at its periphery through necrosis of the cell wall. This cell wall around the spreading area of necrosis, however, is maintained by proliferation of new connective-tissue cells. In this manner the nodules develop and become the size of hemp seed or larger.

Following this development of the lesions many adjacent areas of necrosis may coalesce and finally produce great necrotic nodules the size of a hen's egg.

The infectious organisms may be picked up by the leucocytes and carried into the lymph spaces of the stroma inducing the formation of the new areas of necrosis or daughter nodules.

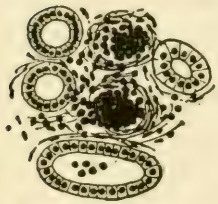

FIG. 5. — Stroma-infection [interstitial mastitis] of chronic character. Pyobacillosis (150). Two groups of cells (emigration) are visible in the swollen cellular intralobular connective tissue. The tubules are compressed. In one tubule a few cells are present. By the use of bacterial stains a few bacteria, pyogenes bacilli, were demonstrated in the centre of the cellular foci.

The necrotic nodules may open a milk-duct and thus induce a secondary milk-infection [parenchymatous mastitis]. In the same way, but less frequently, they may open the skin by ulceration and cause fistulæ.

Because of their position in the stroma the nodules compress the tubules and produce pressure atrophy of their cells. The milk is most altered in quantity which is decreased more or less. The composition is less changed. The fat content lessened. It is poor milk.

Complication by saprophytic invasion is rendered impossible. It is closed off by a completely circumscribing wall of connective-tissue cells.

If a secondary milk-infection is produced by ulceration of a milk-duct or a fistula formed by ulceration of the skin, saprophytic invasion with its subsequent putrid softening is possible through the openings in the necrotic areas in the stroma.

A usual obstacle to such saprophytic invasion, however, is the fact that the organisms which produce the necrosis have a strong attraction for leucocytes. Thus these cells are present in great numbers and rapidly pick up and destroy the saprophytic bacteria. Saprophytic invasion is prevented in this manner.

If, on the other hand, the leucocytes in a secondary infection are repelled by a very strongly virulent infectious organism, a saprophytic invasion is possible and in fact usual.

GENERAL CONDITIONS

Blood-infection. — A blood-infection can occur through ulceration of a vein and through lymph-infection. Since the infectious organisms exert a strong attraction upon the leucocytes the blood is soon cleansed by phagocytosis. The organisms are transported to the lung tissue where secondary metastatic foci may be formed.

Intoxication.—The stroma-infection concerned progresses to a chronic septic-intoxication with indistinct fever symptoms as a rule. It induces a chronic progressive inappetency and emaciation which finally terminates in death.

An unimportant but apparent change is the thickening of the skin (sclerosis) with a rough hair coat. This alteration is probably caused by the direct action of the toxin.

If gangrene be added the intoxication is septic-putrid. The fever is more pronounced and death may be induced in a somewhat acute manner.

Termination.—This type of stroma-infection develops into a chronic progressive destruction of the udder, which can endure for a year. Furthermore it advances to a progressive intoxication with emaciation which can terminate in death.

Summary

I. Ports of infection.
- A. Infection through openings in the udder.
  - 1. Natural openings: the teat canal.
    - The milk as a natural culture medium for bacteria.
    - The causes of sterility of milk in the udder.
      - A. Physical nature.
        - The end opening of the teat canal.
        - The direction of the milk canals.
        - The milk stream.
      - B. Biological nature: the leucocytes.
      - The predisposing factors for infection through the teat canal are:
        - AA. Physical nature.
          - Arrest of milk secretion.
          - Relaxed musculature.
          - Infection with milk tubes and other foreign bodies.
        - BB. Biological nature: virulent infectious organisms.
      - Prophylaxis.
        - Milking time.
        - Cleanliness.
        - Isolation of animals with contagious mastitis.
  - 2. Artificial openings: udder wounds.
    - Defence of wounds against infection: the cell wall, the inflammation.
    - Predisposing factors to infection of the wound.
    - Blood infiltration and necrosis.
- B. Infection by the blood stream.
  - Hypothesis: blood-infection.
  - Causes of blood-infection.
  - Infection of the udder-blood.
  - Occurs,
    - In the most strongly exposed parts of the udder, formation of metastatic nodules.

II. Udder-infection in general.
A. Milk-infection.
Definition.
Ports of infection: primary milk-infection, infection through the teat canal or through wounds.
Secondary milk-infection is one following stroma infection.
1. Local conditions.
A. Primary lesion.
Quantity: circumscribed or diffuse.
Circumscribing factors of a physical and biological nature.
Quality: Superficial necrosis.
Deep necrosis.
Nodular necrosis.
Diffuse necrosis.
Alterations of the milk.
B. Inflammation.
Quantity: circumscribed, diffuse.
Quality: catarrhal-purulent.
Croupous-purulent.
Sequestrating-purulent.
Alterations of the milk.
Complication with saprophytic invasion.
Alterations of the milk.
2. General conditions.
A. Blood-infection.
B. Intoxication.
Septic-intoxication.
Septic-putrid intoxication.
Termination: relative health: seldom death.
B. Stroma-infection.
Definition.
1. Stroma-infection of an acute character.
Ports of infection.
Primary stroma-infection: wounds, seldom blood-infection; secondary stroma-infection: internal wounds following a milk-infection.

A. Local conditions.
Primary lesion.
Inflammation.
Sequestration and necrosis.
Sloughing, softening, encapsulation.
The milk.
Complication with saprophytic invasion.
B. General conditions.
Blood-infection.
Intoxication.
Termination.

2. Stroma-infection of a chronic character.
Ports of infection: teat canal, wound or blood.
Transportation of infectious organisms by leucocytes.
A. Local conditions.
Primary lesion.
Inflammation.
Distribution of progressive necrosis by the leucocytes.
Secondary milk-infection, fistula formation.
Milk.
Complication with saprophytic invasion.
Causes of its infrequent occurrence.
B. General conditions.
AA. Blood-infection.
BB. Intoxication.
Chronic septic-intoxication.
Septic-putrid intoxication.
Termination: progressive destruction of the udder.

The milk-infection and stroma-infection apparently originate together very infrequently, however, as in an acute wound infection.

As a rule they are very sharply separated originally, but may later complicate each other.

Milk-infection often becomes complicated with the stroma-infection, which as a rule is less distinct, but as in nodular necrosis

may become more discernible. Chronic stroma-infection, likewise, becomes complicated with a milk-infection which is indistinct at first but later becomes very severe and pronounced.

The acute primary stroma-infection due to external udder-wounds, however, runs its course without complication, if it does not pass into a milk-infection in the beginning.

The primary milk-infection is the most common, next comes the chronic stroma-infection. The primary acute stroma-infection is comparatively uncommon because of the important port of infection, the udder-wound.

CHAPTER V

TYPES OF INFECTION

UDDER-INFECTIONS may be divided primarily into two groups, depending upon the types of infectious organisms: (1) simple or single infection; (2) complex or mixed infection.

Simple Udder-infection.—The simple udder-infection is caused by one kind of bacteria.

There are, however, different kinds of simple infections because the type of organism which causes one case of simple infection may be of a different type in other cases of the simple infection.

These types of infection induced by the different types of bacteria are divided into two classes:

1. That type of infection which principally shows typical milk-infection and externally only stroma-infection of an acute character.

2. That type of infection which shows a typical stroma-infection of a chronic character.

Types of Infection Which Show Principally Milk-infection or Externally Only Stroma-infection of an Acute Character.—These infections may be caused by three different kinds of bacteria: (*a*) bacteria of the streptococcic group, streptococci; (*b*) bacteria of the staphylococcic group, staphylococci, and (*c*) bacteria of the colon group, colon bacteria or colon bacilli.

The disease is named after the type of organism: Streptomycosis mammæ, staphylomycosis mammæ, and colibacillosis mammæ, or udder-streptomycosis, udder-staphylomycosis and udder-colibacillosis.

Types of Infection Which Show Typical Stroma-infection of a Chronic Character.—(*a*) Bacillus pyogenes; (*b*)

the tubercle bacillus; (*c*) the actinomyces fungus; (*d*) the necrosis bacillus.

They are named after the organism causing the infection: Pyobacillosis mammæ, tuberculosis mammæ, actinomycosis mammæ, and necrobacillosis mammæ, or udder-pyobacillosis, udder-tuberculosis, udder-actinomycosis, and udder-necrobacillosis.

Complex Udder-infection or Mixed Infection.—The mixed infection is caused by two or more types of bacteria.

The bacteria which cause this disease are of the same type that cause the simple infection.

The mixed infection may start originally as a primary mixed infection.

Usually, however, it is originally a simple infection and becomes complex or mixed later through secondary infection (secondary milk-infection).

CHAPTER VI

UDDER-STREPTOMYCOSIS (STREPTOCOCCIC MASTITIS)

THE infectious organism is one or more of the streptococcic group of pathogenic type.

CHARACTER.—The pathogenic streptococcus is a small egg- or spherical-shaped organism which occurs regularly as a chain of two or more individuals joined together. It is easily stained with the common anilin dyes and also with Gram's method. The cultures are light or thin on the usual cultural media, especially surface growths. Gelatin and milk are unaltered.

The last characteristic, the condition of the milk, sharply differentiates the pathogenic streptococci from the non-pathogenic lactic acid streptococcus, which is found in milk, and which resembles the pathogenic streptococcus to such a great extent culturally and morphologically.

There are names for the different types of pathogenic streptococci, as diplococcus pneumoniæ and streptococcus pyogenes, both found in man, streptococcus equi and Schutz diplococcus, both in the horse, and also the streptococcus mastitidis found in the cow.[1]

These groups of streptococci are differentiated principally through their various sources. Otherwise they show very little morphological and biological difference. It is clear that the place in which the organism was found, its source, is no authentic and definite type characteristic, and

[1] The galactococcus fulvus and galactococcus versicolor isolated from mastitic milk by Guillebeau certainly belong to the streptococcic group, while the galactococcus albus, which was only once isolated by Guillebeau, perhaps belongs with the micrococcus tetragenus (sarcina tetragena, Lehmann and Neumann).

the appearance of the pathogenic streptococcus changes considerably under different conditions and therefore presents less effective type characteristics. Special diagnostic type characteristics are not known. For a practical bacteriological classification of the pathogenic streptococci we must wait for further work to be done. [Blake, The Classification of Streptococci, *Journal of Medical Research,* March, 1917.]

Occurrence.—The pathogenic streptococcus is not confined to any special geographical district but appears to be somewhat uniformly distributed. In Sweden it occurs throughout the entire country. Its occurrence is general. The frequency of the infection, especially the wound-infection, indicates that it often occurs in the dust, in the soil of the cow stalls, and in the excrement.

Morphology.—The pathogenic streptococcus presents variations in size. The greatest diameter can vary from 0.9 to 2.3 micra. These are dwarf to giant forms. Usually, however, the greatest diameter is about 1 micron.

The form as a rule is egg- or lancet-shaped, seldom regularly round like a ball. Frequently the egg forms are so affected that they resemble rod-shaped organisms.

As above stated, the individuals form chains. The number of individuals in each chain is variable. In animal bodies the chains are as a rule short, usually only two individuals (diplococci) or from three to five individuals. In body fluids, as milk or pleuritic exudate, however, the chains may reach a considerable length, 100 individuals or more. Upon cultivation, especially in fluid culture media, as bouillon, the chains are usually very long. With the diplococci and the short chains the individuals are arranged longitudinally like eggs with the points against each other. In the longer chains the individuals are usually arranged transversely as eggs with their long sides against each other. The explanation of this condition is that in the latter case

the division is so rapid that the full length is never reached and thus through dividing the breadth of the cell is greater than its length.

In animal bodies the organism frequently presents a wide capsule. The capsule appears to occur principally on the short chains, especially the diplococci type.

The capsule formation is most distinct in mice (in the liquid of the spleen). In the other laboratory animals and in our domesticated animals it is indistinct as a rule. In man it is very distinct (diplococcus pneumoniæ). It is visible after staining with carbol-methylene blue or with Gram's method (Fig. 6).

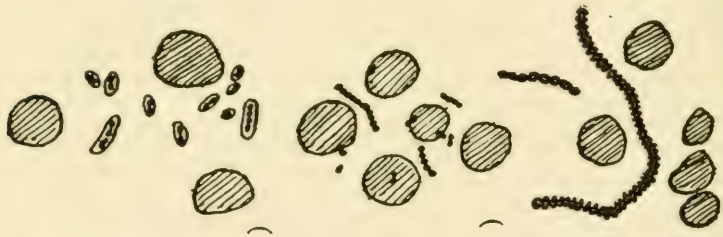

Fig. 6.—The pathogenic streptococci in milk sediment. Gram. (150). Three different types.

The streptococcus is easily stained with the common anilin dyes as well as by Gram's method, in which decolorization with alcohol need not be long, however. It is less Gram positive than the staphylococcus, but more so than the bacillus pyogenes. The streptococcus is immotile.

Cultivation.—It grows best at body temperature (ca. 38° C.) and appears not to grow under 18° C.

It grows aërobically and anaërobically but inclines more toward the anaërobic tendency.

In order to isolate streptococci in pure culture, it is usually necessary that the culture medium contain serum. If one has first cultivated it on this medium it is easier to grow it on media without serum.

For the purpose of isolation I pour plates of serum-

gelatin-agar (gelatin 5 per cent., agar 0.75 per cent., and serum 15 per cent., the latter is added shortly before pouring). This medium is very soft and the colonies maintain a characteristic form. After one day at 38° C. the colonies manifest small, grayish-white visible points, and after a few days a few sparse colonies in the last plate reach the size of ca. half a pinhead. These distinct colonies are deep colonies. Under the microscope they are round or oval and as a rule have an uneven, jagged or finely fringed margin which is infrequently even (Fig. 7). The colonies which reach the surface show a round, bluish-gray, thin, barely visible membranous surface growth the size of a hemp seed. These surface colonies are sparsely present and may be absent altogether.

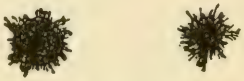

Fig. 7.—Colonies of streptococci. Deep colonies. Serum-gelatin-agar plates. T. 38° C. 3 days. (× 30).

With stab cultures in gelatin-agar, agar (38°) and gelatin (20°), a grayish-white granular filament forms along the line of puncture and as a rule there is no distinct surface growth (Fig. 8). The gelatin is not liquefied. Growth at 20° C. is slow.

Fig. 8.—Culture of streptococci. Stab in serum-gelatin-agar. T. 38° C. 3 days.

With streaks upon agar, coagulated serum (38°) and gelatin (20°) small, fine grayish-blue colonies like drops of moisture form along the streak and then coalesce, producing a fine thin filament.

Upon potato (38°) the streptococcus grows slowly without forming colonies that are visible to the naked eye.

In bouillon the streptococci form grayish-white bacterial granules or colonies the size of a pinhead on the bottom and along the side of the test tube. The media does not become turbid. Upon shaking the test tube the colonies are destroyed and the bouillon is made uniformly turbid.

In milk bacteria grow without changing the medium.[2]

In sugar media (glucose, maltose, lactose, saccharose) no gas but a slight quantity of acid is formed so that the media can contain 1–2 per cent. normal acid.

Resistancy.—Streptococci are very sensitive. Usually daylight will destroy a culture in a day. In stab cultures or in blood in hermetically sealed capillary pipettes, kept from the light, the organism may live for 3–4 months. Highly virulent streptococci appear to be more sensitive and less vigorous than those of less virulence.

Pathogenicity.—The virulence fluctuates greatly. There are pathogenic streptococci which produce no lesions in experimental animals, others which induce local lesions only, and still others which cause death in variable lengths of time. The virulence can be raised by passage through animals.[3]

Mice are the most susceptible to the infection. One drop of bouillon culture subcutaneously can kill a mouse in from 1 to 7 days or sometimes longer, 14 to 21 days, depending upon the virulence.

Autopsy.—If death occurs suddenly one perceives a slight spleen swelling, and the point of inoculation is either

[2] As a rule, the literature states that the pathogenic streptococci coagulate milk quickly. This is insistently stated, especially with reference to mastitic streptococci. Of nine different strains of streptococci which I studied in this respect, of which seven were mastitic streptococci, only one strain coagulated milk. This strain was isolated in summer from a one-day-old milk sample; I had undoubtedly isolated the lactic acid streptococcus instead of a pathogenic streptococcus.

The mistake in the literature mentioned probably rests upon this fact, since the lactic acid streptococcus is a very common saprophyte.

[3] The literature states that mastitic streptococci are never pathogenic for small experimental animals, which is often, but not always, the case.

unaltered or shows an œdematous swelling. If it runs a longer course the spleen is very large, and the liver may show small multiple areas of necrosis or abscesses and the point of inoculation, local necrosis with abscess formation.

The animals usually die of bacteræmia, although they may also die of septic intoxication without bacteræmia.

In bacteræmia the number of streptococci is small but they are comparatively numerous in the spleen pulp.

Dogs.—These animals are also quite susceptible to the infection. One c.c. of a bouillon culture injected subcutaneously can kill the animal in one to five days, depending upon the virulence.

Autopsy.—If death occurs very suddenly the cadaver shows spleen swelling, serous extravasation into the abdominal and thoracic cavities and into the pericardial sac, lung œdema and a local œdematous swelling at the point of inoculation. If the course be more protracted we may find a serofibrinous peritonitis, pleurisy and (or) pericarditis and not infrequently serofibrinous arthritis and pneumonia.

As a rule the animals die of bacteræmia. Great numbers of bacteria are found in the spleen pulp. If they do not die of bacteræmia the organisms are found in great numbers in local nodules.

Pigeons are somewhat resistant to the infection. They may be killed however in from one to two days by intramuscular injection of one c.c. of a highly virulent culture.

Autopsy.—A serous exudate is found in the abdominal cavity and the pericardial sac. The blood is rich in streptococci.

Guinea-pigs are very resistant to the infection. One c.c. of a highly virulent culture injected subcutaneously in a guinea pig only produces local necrosis at the point of inoculation with abscess formation. The animal does not die.

Cows.—No. 1: Fourteen days after parturition a milch cow was injected in one teat with 10 c.c. of a very virulent bouillon culture of streptococci which had been isolated from a case of mastitis. The culture was controlled for pathogenicity on two mice, one of which died in one day, the other in two days.

On the day after the infection the body temperature was 41.1° C. (ca. 106° F.). The cow lay down, sweat, and refused to eat. The infected quarter was greatly swollen and painful to pressure. The skin was tight and doughy to the touch. The milk of the quarter was diminished in quantity and presented a yellowish purulent appearance. Microscopically it was rich in streptococci.

Two days after the infection the fever had subsided. The appearance of the quarter was the same. The milk of the quarter was still less in quantity and consisted of a yellowish turbid serum plus yellow fibrin clumps. Numerous streptococci were found in the fibrin. Five days after the infection the swelling of the quarter was greatly increased, stone hard and somewhat less sensitive. The quantity of milk from the quarter was insignificant. The milk was purulent and consisted of yellow turbid serum plus yellow clumps of fibrin which contained few streptococci.

One month after the infection this quarter and its milk showed the same appearance. The animal was then slaughtered.

Autopsy.—The infected quarter was firm and very much swollen. The incision was somewhat dry (absence of milk) and the cut surface was smooth, yellowish-gray and firm. The lobuli were small and showed no milk points (cross sections of fresh tubuli). Upon pressure yellow cylinders of fibrin were expressed. In addition five necrotic nodules were found which were about the size of a walnut, rounded, sharply circumscribed, firm and surrounded by

pus and a thick wall of connective tissue. The walls of the milk-ducts were thickened.

No. 2: Three weeks after parturition this cow was inoculated in one teat with 10 c.c. of a bouillon culture of streptococci of less virulence, which was also isolated from a case of mastitis and was controlled on two mice, only one of which died in one day.

On the day after the infection the body temperature was 39.7° C. (ca. 103.5° F.). The infected quarter of the udder was not noticeably changed. Its milk was of normal appearance but gave a rich yellowish-white purulent sediment which contained few streptococci.

Two days after the infection the animal was free from fever. The quarter was not visibly altered. Its milk was thick, yellowish-white and gave a rich sediment of yellowish-white pus containing few streptococci.

Three days after the infection the quarter produced decidedly less milk, which was yellowish-white, thick and clumpy. In other respects it was the same as before.

Five days after the infection the milk of the quarter was yellowish-gray and flocculent, otherwise the same.

Six days after the infection the quarter was somewhat firmer. It gave a small quantity of milk which was yellowish-gray, thick as pulp, puslike and contained but few streptococci.

Fourteen days after the inoculation the infected quarter and its milk showed the same continued alteration. The animal was then slaughtered.

Autopsy.—The infected quarter was not markedly swollen, but thick and firm. The milk was yellowish-gray, puslike and thick as pulp. The incision was smeared with the same kind of thick pus. The cut surface was even, yellowish-gray and firm in its lowest part. The lobuli showed no milk points. Upon pressure yellow-gray fibrin plugs were expressed.

Udder-infection

The streptococcus causes milk-infection and acute stroma-infection. The milk-infection is the most important.

milk-infection [*Parenchymatous Mastitis*]

This is almost always primary, very seldom secondary. The primary milk-infection as a rule is caused by infection through the teat canal, seldom through the rupture of tubuli in fresh wounds. The secondary milk-infection is one following the embolic stroma, infection. It is very infrequent indeed.

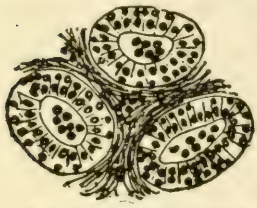

Fig. 9.—Typical chronic streptomycosis. Milk infection [parenchymatous mastitis] (× 150). Epithelial layer double. The epithelial cells show weak nuclear staining due to degeneration. Intralobular connective tissue thickened. Tubules compressed. Cells in the intralobular connective tissue and within the tubules. A moderate number of streptococci were demonstrated in the tubules with bacterial stains.

The local condition is originally confined to one quarter, as is the usual case, but later other quarters may become infected.

The primary infection varies considerably in quantity and quality, depending upon the virulence of the infectious organism and the condition of the udder (time after calving). Nodular necrosis can be produced, although I have never seen diffuse necrosis in this infection.

The quantity and quality of the inflammation varies with that of the primary lesion.

Since the infectious organisms cannot liquefy the coagulation necrosis, the exudate often shows a decidedly croupous character.

An infectious organism of low virulence which only produces a circumscribed inflammation in the parts around the base of the teats can easily induce a chronic progressive condition. The quantity of milk is only slightly diminished by this condition, which is of little importance in the beginning. It is, however, a sufficiently good culture medium

for the infectious organisms present. Only the number of cells is slightly increased, due to the low grade inflammation. In consequence of this the organisms multiply, enter new tubules and lobuli, extending the inflammation and from here they can spread the condition further upward and outward (typical chronic streptomycosis).

A secondary infection frequently occurs. Colon bacilli and staphylococci may complicate the condition. Tuberculosis and pyobacillosis may also be induced, although pyobacillosis is the most common secondary infection.

In these secondary infections the new infection may conceal the primary infection with its own individual and aggressive character.

A saprophytic invasion of an original streptococcic infection is infrequent and only occurs in those of the highest streptococcic virulence when the cell resistance has been lowered.

GENERAL CONDITIONS

Blood infection can occur and manifest itself clinically by multiple arthritis, which is common in the hock joint. Fever can be more or less severe or be absent, depending upon the virulence of the organism and the condition of the udder. It is diminished by the inflammation so that the animal is free from fever after a few days, as a rule. Fever is seldom fatal.

In saprophytic invasion with gangrene, fever becomes chronic and death may occur.

Termination.—The condition is seldom fatal. As a rule it runs a course of from a few days to months and years to recovery. The udder usually heals with diffuse sclerosis and parenchymatous atrophy, which conditions are most severe following gangrene.

Clinical Symptoms.—We discriminate here between the acute and chronic condition.

The course of the acute condition varies from a few

days to months. One quarter, seldom more, is affected. The quarter rapidly becomes greatly swollen, hard and painful following a severe primary lesion, or soft, flabby and somewhat collapsed following a less severe primary lesion.

The quantity of the milk is greatly diminished, and the milk is thick, yellow, puslike and often contains viscid flakes of fibrin. In gangrene the milk is homogeneous and stinking.

Upon microscopic examination of the sediment one finds very few Gram positive cocci of oval shape and arranged as diplococci or perhaps in short chains.

In the first few days and also later the animal may show fever if gangrene has set in.

The course of the chronic condition extends from several months to a year.

From a clinical point of view this chronic streptomycosis can be divided into an atypical chronic and a typical chronic streptomycosis.

ATYPICAL CHRONIC STREPTOMYCOSIS

This begins suddenly as an acute inflammation, although the inflammation is not, or at least not completely, high grade but becomes chronic.

One or more quarters are affected. The quarter is usually moderately swollen and very firm. Not infrequently one feels hard necrotic nodules in the quarter. The milk is more extensively purulent and consists of a turbid yellow fluid plus viscid fibrin flakes, or on the other hand, it may present a comparatively normal appearance, but is diminished in quantity and contains a more or less great number of fibrin flakes.

Upon microscopic examination of the sediment one usually finds a few oval or lancet-shaped Gram positive streptococci arranged as diplococci or in short chains.

Fever occurs in the early stages but later subsides unless gangrene sets in, in which case the fever becomes chronic.

This begins imperceptibly and proceeds insidiously. The alteration of the udder and the milk takes place by slow degrees.

One or more quarters become involved. The quarter is not enlarged, but often diminished in size and after milking does not collapse at all or only slightly reduces. That portion of the udder around the base of the teats, especially the parts anterior to the teats of the fore quarters and posterior to the teats of the hind quarters, appear, after milking, to continue to be filled with milk and to be hard to the touch or sclerotic.

These pathologically altered parts increase in area by extending forward, backward and slowly upward.

The quantity of milk decreases by slow degrees. The milk is at first imperceptibly but later always distinctly changed. It coagulates on boiling because of the albumin contained. It is thicker, almost like cream, due to the increased number of cells, and can contain blood at the same time. Finally it becomes decidedly puslike and consists almost entirely of cells.

Upon microscopical examination of the sediment one finds a great number of Gram positive streptococci in long chains. The condition does not present the symptom fever.

Prognosis.—The condition is seldom fatal. High fever, especially when gangrene is a complication, is very dangerous.

The infection certainly diminishes the functional capacity of the diseased quarter more or less.

Following an acute infection of short duration, however, the functional capacity completely returns to normal, due to the insignificance of sclerosis.

The chronic infection tends toward complete destruction of function. The parenchyma of the quarter under-

goes severe atrophy following pressure of the interstitial connective tissue, which undergoes a high grade of proliferation with induration or sclerosis of the part.

Treatment.—*Prophylaxis.*—The danger of infection in these cases is a very great one. Udder-streptomycosis, especially the acute type, can pass through a cow stable like a high-grade enzoötic disease. Therefore, isolate the diseased cow from the others and milk her last, and of the quarters of the udder, milk the diseased one last of all. Do not draw the infected milk upon the floor, but into a receptacle of disinfectant with the idea of preventing the further spread of the infection.

The treatment proper is divided into treatment of the general condition and treatment of the local condition.

Treatment of the General Condition.—Fever is treated with febrifuges. Camphor, five grains daily, is especially recommended, because it is excreted with the milk and can thus bring about local disinfection. If the milk of the other quarters is to be used, some other febrifuge must be employed, such as antifebrin (acetanilid), which does not impart its taste to the milk.

If the impaction secondary to fever is severe a purgative of Glauber's salts is given.

Treatment of Local Condition.—One should milk out the infected quarter carefully. Then inject from 300 to 500 c.c. of lukewarm, previously boiled 4 per cent. solution of boric acid, which exerts a deleterious effect upon the growth of the bacteria without too much irritation to the udder tissue. This solution can remain in the udder without danger and can be milked out in about four hours (Bigoteau).

Externally one can use a mild skin irritant, as concentrated iodine solution, rubbed on daily for a day or two. This stimulates an acute counter inflammation and resorption.

The udder should be milked carefully twice a day, but not more frequently, as the udder should rest.

If the condition is obstinate and threatens to become chronic one can cause the milk secretion to diminish by inflation with air and thus inhibit the growth of the bacteria. One can also combine the boric acid injection with air-inflation. If this does not help there is nothing left but to cause the udder to go dry. When it is possible, only the diseased quarter is induced to enter the non-lactating state.

In cases of severe fever following gangrene one can prepare an easy path into the gangrenous mass by cutting off the teat,[4] then induce healing and sclerosis by deep point-firing or deep cautery into the quarter.

In nodular necrosis the course can be shortened by operative removal of the nodules (sequesters).

STROMA-INFECTION

This is somewhat infrequent and of less interest.

The wound-infection is a complication of udder wounds. It presents local necrosis with subsequent suppuration and proceeds with or without fever.

Secondary infection with staphylococci and colon bacilli are common and gangrene may also be produced.

Wound-infections are treated following the usual surgical methods.

Therefore one provides free drainage for the wound secretion, which condition diminishes the nutrient fluid

[4] This treatment is somewhat dangerous, however, if the diseased cow be permitted to remain with the other cows, since after the operation the quarter is continuously discharging infected milk, which spreads the infectious organisms and may be a source of infection to the healthy cows. Therefore the diseased animal must be well isolated after the operation and her stall must frequently be disinfected.

media for the bacteria. Necrotic parts are removed early as a prophylaxis against gangrene. The wounds are cleansed once or twice a day with a mild antiseptic.

As the development of inflammation is delayed by severe primary lesions, so wounds may be treated by inflammatory irritants, as iodine solution, or with cautery. Both of these methods are of a disinfecting character.

In fever febrifuges are indicated.

An embolic stroma-infection may perhaps occur. It is undoubtedly rare, however.

The primary condition inducing the blood infection is probably in this case a streptococcic endocarditis, at least such is the case in most instances. In one case of infectious endocarditis that I observed the cow showed evidences of streptomycosis. Streptococcic endocarditis appears to be quite rare, however.

Following penetration of the necrotic tubuli by the primary lesion, *i.e.*, by the embolic nodule, or following ulceration of the tubule, a secondary infection is induced.

It probably appears clinically as a suddenly occurring milk-infection (parenchymatous mastitis) affecting one or more quarters but usually one or both hind quarters.

Summary

I. Infectious organism: the pathogenic streptococcus.
   Character.
   Occurrence.
   Morphology.
   Cultivation.
   Resistance.
   Pathogenicity.

II. Udder-infection. Udder-streptomycosis.
   A. Milk-infection [parenchymatous mastitis].
      Local conditions.
      General conditions.
      Termination.

Clinical symptoms.
Acute streptomycosis.
Chronic streptomycosis.
Atypical chronic streptomycosis.
Typical chronic streptomycosis.
Prognosis.
Treatment.

B. Stroma infection [interstitial mastitis].
Wound-infection.
Embolic stroma-infection.

CHAPTER VII

UDDER-STAPHYLOMYCOSIS

The Infectious Organism

The infectious organism is the pathogenic staphylococcus pyogenes.

The species name pyogenes which means pus producing is becoming obsolete since the pathogenic staphylococcus can cause necrosis without suppuration.

Characteristics.—The pathogenic staphylococcus is a small round microörganism which appears alone or in groups of two or more, or presents square or irregularly formed groups of individuals. They are easily stained with the common anilin dyes and after Gram's method. Their growth on culture media is vigorous, especially the surface growths. It liquefies gelatin and coagulates milk.

Occurrence.—This coccus is similar to the streptococcus in its geographical distribution and is present everywhere in Sweden. It is very common and appears frequently in the dust of cow stables.

Morphology.—The size of the pathogenic staphylococcus is somewhat variable. There are large and small cocci. As a rule it is one micron in diameter and somewhat regularly globular in shape. It frequently occurs as a single coccus or monococcus and also joined in groups. These groups consist of two (diplococci) or more individuals, such as a four-sided group consisting of from four to six individuals, or as an irregularly shaped group simulating the arrangement of a bunch of grapes. These groups consist of from 20 to 100 or more individuals.

Not infrequently these cocci may also be arranged in a line forming a chain, which is, however, short. The mono- and diplococci occur in the greatest numbers, while the

groups are less frequent and may be absent altogether (Fig. 10).

As previously stated, they are easily stained with the common anilin dyes and following the method of Gram. They are immotile and do not form a capsule.

Cultivation.—The staphylococcus grows best at body temperature (ca. 38° C.). It also grows well at room temperature. It grows aërobically and anaërobically, but somewhat better aërobically, however. It can be cultivated on the common media and grows well without serum.

In the serum-gelatin-agar plates colony formation is distinct after one day at 38° C. After two days the deep

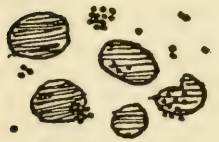

Fig. 10.—Pathogenic staphylococci in croupous membrane from udder. Gram. (× 550).

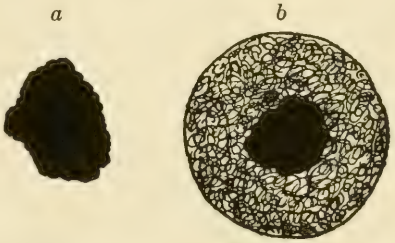

Fig. 11.—Colonies of staphylococci. *a*. Deep colonies. *b*. Superficial colonies. Serum-gelatin-agar plates. T. 38° C., 2 days (30).

colonies in the last plate appear as sparse pinhead size whitish-yellow, round or oval points.

Upon microscopic examination with 30 diameters enlargement they are opaque (black), coarsely granular and in consequence of this granularity show a crenated margin. Surface colonies are numerous and form porcelain-white or yellow circular or at times irregularly formed, elevated knob-shaped growths ca. 6 mm. in diameter (Fig. 11).

The staphylococcus frequently produces a yellow pigment by which the colonies, especially the surface colonies, become a distinct yellow color. We differentiate the white and yellow staphylococcus. The white staphylococcus occurs more frequently than the yellow in our domesticated animals.

In stab cultures in gelatin-agar and agar at 38° C. it forms a nail-like growth (Fig. 12). The nail-like growth in the stab is wedge-shaped and most vigorous near the top. The head of the nail or surface growth is thick and glistening and covers the greater part of the surface. In gelatin stab culture at 20° C. the growth is similar, the medium is liquefied more or less quickly (ca. 3 days) in goblet or funnel shape (Fig. 13). The liquefied medium is diffusely turbid and has a dense bacterial deposit on the floor against the unliquefied medium, but at the expense of the surface growth.

In streak cultures upon agar, potato and coagulated blood serum at 38° C. they form elevated beaded round colonies which coalesce and produce a thick band along the streak.

Fig. 12.

Fig. 13.

Fig. 12.—Culture of staphylococci. Stab in serum-gelatin-agar. T. 38° C., 3 days.

Fig. 13.—Culture of staphylococci. Stab in gelatin. T. 20° C., 10 days. Horn-shaped liquefaction.

In bouillon the organism grows with diffuse turbidity of the medium and forms dense bacterial deposits on the bottom of the tube. It does not present a surface growth.

Milk at 38° C. is usually somewhat slowly coagulated, a condition which endures for from several days to a week, usually three to five days.

In media which contain sugar, *i.e.*, glucose, maltose, lactose, and saccharose, it forms acid but no gas, so that the medium finally contains ca. 2 per cent. normal acid.

Resistancy.—The staphylococcus does not form spores, although it is very resistant. It resists light and drying in a sealed culture tube for a year or probably longer. It endures daylight very well.

Pathogenicity.—The virulence varies greatly. There are pathogenic staphylococci which produce no alterations when injected subcutaneously, others which only cause local necrosis and abscess formation and again others which cause death rather quickly.

I have not seen bacteræmia in laboratory animals, although it may be produced in dogs. At the point of inoculation, however, the organism can be found.

Autopsy of cases which have run an acute course shows only an œdematous swelling at the point of inoculation and no distinct lesions elsewhere.

Mice, dogs and pigeons may be killed in one day by an organism of high virulence.

Cows.—Twelve c.c. of a one-day-old culture of staphylococci which had previously been isolated from a severe case of mastitis was injected into one teat of a milch cow about six months after parturition. This staphylococcus had been preserved in culture for sixteen months sealed with paraffin and kept from the light. An abscess was produced at the point of inoculation in a control mouse.

Before inoculation the quarter of the udder showed no alterations. The milk was of normal appearance but less in quantity and showed a yellow sediment consisting of a few pus cells and streptococci.

Four hours after inoculation the body temperature was 39.1° C. [102.2° F.].

On the day following the infection the animal was free from fever. The quarter was moderately swollen and was more firm to the touch than usual, but not painful. The quantity of milk was reduced and the milk yellow and thick and contained a copious sediment composed of yellow pus, staphylococci in great numbers and some streptococci.

Two days after the inoculation the milk was yellow and thick and contained a moderate amount of sediment consisting of yellow pus and frequent chains of cocci. It was otherwise the same.

Three days following infection the milk was whitish-gray and flocculent and presented a moderate quantity of sediment consisting of yellow, fibrinous pus and a moderate

number of chains of cocci. Other changes were the same as above described.

Four days after the infection the consistency of the quarter was the same as before inoculation. The milk showed a normal appearance again and a lessened quantity of sediment, which consisted of pus and a moderate number of chains of cocci.

Six days after infection this condition remained unchanged. Upon cultivation of the sediment of the milk on serum-gelatin-agar plates a moderate number of streptococci but no staphylococci were found.

Thirteen days after the inoculation the condition remained unchanged and the animal was slaughtered.

Autopsy.—The infected quarter was slightly swollen and firm. The incision was juicy with milk. The lower part of the cut surface was even and uniform, grayish-white and yellow. The lobuli here present no milk points. Upon pressure here and there small yellow fibrin cylinders were expressed (chronic streptomycosis).

The virulence of the staphylococcus was obviously not high and the infection did no more to favor drying of the quarter than did the subsequent streptococcic sclerosis. Therefore I only obtained a mild infection of the udder which reached its height as a catarrhal inflammation in four days. The chronic streptococcic infection was obviously favored by this short secondary infection.

Udder-infection

The pathogenic staphylococcus causes milk-infection and acute stroma-infection. The milk-infection is the most important.

milk-infection [*Parenchymatous Mastitis*]

This is always primary and as a rule is caused by infection through the teat canal, seldom by infection through fresh wounds.

The local condition as a rule is confined to one quarter but may, however, be extended to more quarters, especially by highly virulent organisms.

The primary lesion varies with the virulence of the organism and the condition of the udder. Shortly after parturition, especially, a primary lesion can develop into diffuse necrosis of the infected quarter.

In this manner the infection manifests its relation with Nocard's staphylomycosis of sheep: "Mammite gangreneuse de la brebis."

The quantity and quality of the inflammation varies with the primary lesion. The infection appears to have no tendency toward chronicity.

Secondary infection may occur in the form of streptomycosis, colibacillosis, pyobacillosis and tuberculosis.

These secondary infections are marked as a rule by the predominant staphylococci. Later, where the staphylococcus has completely run its course, the condition can be continued as a chronic infection as streptomycosis, pyobacillosis, or tuberculosis.

A saprophytic invasion is common following a severe primary lesion, but otherwise infrequent.

The General Condition.—Infection of the blood appears to occur very infrequently.

The intoxication varies with the virulence of the organism and the condition of the udder, *i.e.*, with the quality and quantity of the primary lesion.

The fatal septic intoxication may easily follow diffuse necrosis of the infected quarter, especially if gangrene develops.

Termination.—The condition may be fatal. The course is always acute and varies from a few days to three weeks. If the infection is removed by the inflammation the condition usually develops into a low-grade sclerosis.

Clinical Symptoms.—The condition is always acute,

as previously stated. The quarter is more or less severely swollen and hard. The quantity of milk is more or less diminished. The milk may contain a red-colored serous fluid, indicating a primary lesion of diffuse necrosis, or consist of a yellow, thick, purulent fluid indicative of inflammation and which may contain free yellow croupous membranes in severe udder conditions.

Upon microscopical examination of the sediment one observes Gram positive staphylococci as mono- or diplococci but seldom in groups.

When the primary lesion is severe the condition is accompanied by fever, which is very high if gangrene sets in. When the inflammation begins fever disappears.

Prognosis.—In recently fresh and good milking cows the prognosis is less favorable. A severe primary lesion without indications of inflammation (emigration of leucocytes), but with high fever, is a very unfavorable symptom and is indicative of a fatal termination. Gangrene is also a very unfavorable sign.

Inflammation with purulent milk and reduction of fever is a very good indication.

Treatment.—*Prophylaxis.*—The condition may be entirely infectious. Therefore one should observe the common measures for prevention of infection. One should be particularly careful to see that cows which have recently freshened do not come in direct or indirect contact with the patients through the milking process.

The actual treatment is principally the same for udder-streptomycosis.

In cases which present a severe primary lesion without inflammation but with high fever one may endeavor to save the life of the animal by extirpation of that half of the udder which contains the diseased quarter.

If one does not favor this operation he may apply deep cautery or deep point-firing into the quarter.

Amputation of the teat before gangrene has set in is not to be recommended, since saprophytic invasion is thus made easier and this dangerous complication encouraged.

In abscess formation one may shorten the course of the affection by incising the abscesses which are accessible for the operation.

STROMA-INFECTION [*Interstitial Mastitis*]

The stroma-infection only occurs as a wound-infection and is therefore a complication of an udder wound.

It is a comparatively harmless condition as long as it does not become complicated with a milk-infection.

Septic-intoxication does not take place so readily because of the lack of nutrient media for the bacteria.

It progresses to localized wound necrosis with suppuration. Secondary infection with streptomycosis and colibacillosis is not uncommon, and gangrene may also develop.

This wound-infection is treated following the common surgical methods.

SUMMARY

I. Infectious organism: the pathogenic staphylococcus.
Characteristics.
Occurrence.
Morphology.
Cultivation.
Resistancy.
Pathogenicity.
II. Udder-infection. Udder-staphylomycosis.
A. Milk-infection [parenchymatous mastitis].
Local conditions.
General conditions.
Termination.
Clinical symptoms.
Prognosis.
Treatment.
B. Stroma-infection [interstitial mastitis].
Wound-infection.

CHAPTER VIII

UDDER COLIBACILLOSIS

Infectious Organism

The infectious organism is the colon bacillus or bacillus coli. The name coli is taken from its original habitat, the contents of the colon of man.

The mastitis bacillus described by Kitt as the bacillus phlegmasiæ uberis may be briefly reviewed as the colon bacillus. Kitt's bacillus should have only one flagellum while the genuine colon bacillus should have from four to six flagella. We have so little practical knowledge concerning the flagella of the bacilli that we should venture to distinguish new types only upon the difference in their number.

Characteristics.—The colon bacillus is a short thick rod with rounded ends. It is easily stained with the common anilin dyes, but is not stained after Gram's method. The growth in the common culture media is vigorous, especially surface growths. It does not liquefy gelatin. It coagulates milk with gas production. It splits sugars, as glucose, maltose, lactose, and also often saccharose with the production of gas and acid.

Occurrence.—The colon bacillus, as is the case with the other organisms, is of uniform geographical distribution and occurs everywhere in Sweden. It is a common intestinal parasite and therefore occurs in manure and in the dirt and dust of cow stables.

Morphology.—The size of the colon bacillus varies considerably. There are large and small colon bacilli. The longer vary between one and four microns; only in exceptional cases it is longer, at which times it may reach eight microns. Its thickness varies between 0.5 to 1.5 microns.

As a rule the size of the bacillus is regularly two to three microns long.

The vegetative colon bacillus forms a distinct but, in comparison with its thickness, somewhat short rod with rounded ends. It seldom reaches the unusual length of eight microns, at which times it simulates a short thread. Besides these vegetative specimens one always finds a number of shorter specimens which have an oval form and many of which are also globular. The short forms are most numerous in culture; they are less numerous in the animal body where the vegetative specimens are present in the greatest number.

Fig. 14.—Colon bacilli in milk sediment. Carbolmethylene blue. (× 150.) Two different cases.

The colon bacillus occurs mostly as a single bacillus, but is also arranged in lines consisting of two individuals or diplobacilli, infrequently of three individuals or streptobacilli (Fig. 14).

They are easily stained by the common anilin dyes, but are quickly and easily decolorized following Gram's method.

In preparations from animal bodies it is surrounded by a bright halo like a capsule. This is perhaps an effect produced by the flagella.

The colon bacillus is motile or immotile.[1]

Cultivation.—It grows best at body temperature (38° C.), it also grows well at room temperature. It grows

[1] I classify the immotile bacterium lactis aërogenes (synonym: b. acidi lactici) with the colon bacillus. The real colon bacillus is motile, b. lactis aërogenes is immotile; but in other morphological and cultural characteristics these bacteria are very similar. The real colon bacillus often forms "wander zones" in culture, which formations are not produced by b. aërogenes. Both of these bacteria produce mastitis.

aërobically and anaërobically, but better aërobically. It also grows well on the common culture media without serum.

In serum-gelatin-agar plates at 38° C. the growth is distinct after one day. After two days sparse deep colonies form in the last plate which are the size of a pinhead,

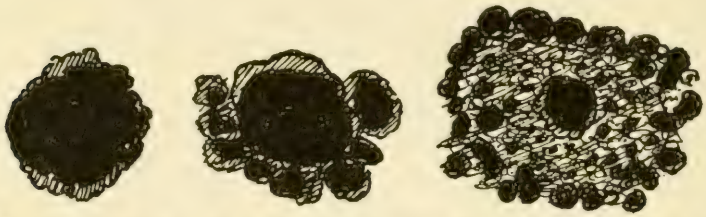

Fig. 15.—Deep colonies of colon bacilli with and without daughter colonies. Serum-gelatin-agar plates. T. 38° C., 2 days (× 30).

yellowish-white, round points which are not infrequently surrounded by one or more broad, grayish-white zones. This zone is produced in the culture media by the bacilli which wander out from the colony. The zone can therefore be called the "wander zone."

Under the microscope the colonies are almost transparent, yellow, round or rounded and have an even or coarsely crenated margin.

Fig. 16.—Surface colony of colon bacilli. Serum-gelatin-agar plates. T. 38° C., 2 days (× 10).

The colonies with "wander zones" show a large colony in the middle, the mother colony, which is surrounded by a more or less broad ring of peripheral or daughter colonies. These colonies present growing margins depending upon the outwandering of bacilli (Fig. 15).

The surface colonies are numerous and form bright gray round or irregularly lobed thin coverings from 6 to 10 mm. Under the microscope they often show a granular centre which depends upon the formation of the colonies in the media (Fig. 16). In stab cultures in gelatin-agar (Fig. 17), agar at 38° C., and gelatin at 20° C. the colonies form

a nail culture. The nail is wedge-shaped, most vigorous above and often has a fine felt-like contour following the formation of daughter colonies in the media by outwandering bacilli (Fig. 18). In soft media like gelatin-agar the nail-like growth is often surrounded by a whitish-gray more or less thick cylindrical zone (wander zone), and the nail-shaped growth in the track of the stab, the stab growth, can become indistinct by a vigorous outwandering of bacilli. The nail head is made of a grayish-white somewhat thin surface membrane which at 38° C. completely covers the surface and in gelatin at 20° C. is small and only covers part of the surface. Gelatin is not liquefied.

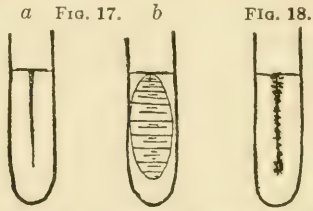

Fig. 17.—Cultures of colon bacilli. Stab in serum-gelatin-agar. T. 38° C., 3 days. Two different strains, *a*, without and *b*, with "wander zone."

Fig. 18.—Culture of colon bacilli. Stab in gelatin. T. 20° C., 6 days. With downy hair-like peripheral outgrowth.

In streaks upon agar at 38° C. it forms whitish-gray round colonies, which coalesce and produce a gray band along the streak. In bouillon and peptone water at 38° C. the colon bacillus grows with diffuse turbidity of the culture media and formation of distinct bacterial deposits on the bottom of the test tube. As a rule it does not form a membrane on the surface, and if it does it is very thin and small.

The colon bacillus coagulates milk somewhat quickly, usually in one day, at 38° C., with the production of gas.

It splits the sugar, glucose, maltose, lactose, and often saccharose with formation of gas and acid so that the medium finally contains 2 to 3 per cent. normal acid. The splitting of saccharose is slow and does not occur at all with some colon bacilli.

The colon bacillus produces more or less indol in peptone water (peptone Witte 3 per cent., sodium chloride 0.5 per cent.) in about six days at 38° C.

The colon cultures have a strong, unpleasant, burnt odor.

Resistancy.—The colon bacillus does not form spores but is nevertheless very resistant. It can live a year in a culture kept in the dark and sealed to prevent drying.

Pathogenicity.—The virulence is very variable. In subcutaneous inoculations of laboratory animals certain colon bacilli manifest no alterations. Others produce only a local necrosis with abscess formation, while others may kill the animal more or less quickly.

As a rule, laboratory animals die of bacteræmia.

On autopsy one finds a more or less enlarged spleen, and the point of inoculation in the subcutis is œdematously swollen or manifests necrosis with inflammation in the surrounding tissue. There are no other distinct lesions.

Mice are very susceptible to the infection. They may die in from one to twelve days. As a rule they die in about three days, seldom after a course of a week or more.

A dog inoculated subcutaneously with a virulent culture did not die but showed a necrotic nodule at the point of inoculation.

A guinea-pig was inoculated subcutaneously with a virulent culture and showed a widespread severe œdematous swelling at the point of injection and also bacteræmia.

A pigeon inoculated in the pectoral muscle with a virulent culture did not die but developed a necrotic nodule at the point of injection.

Cows.—About six months after calving a cow was injected in one teat with 10 c.c. of a one-day-old bouillon culture of colon bacillus which had previously been isolated from a case of mastitis. Of the two control mice one died in two and the other in three days.

Examination of the quarter before infection showed no alterations. The milk was of normal appearance but contained a small quantity of sediment consisting of yellow pus which contained a few streptococci.

Five hours after the infection the body temperature was 39.1° C. (102.2° F.).

On the day after the infection the appetite was diminished and the animal showed constipation. The infected quarter was swollen, firm, red, warm and painful. The quantity of milk was diminished. The reaction of the milk was alkaline. It was yellow, thick and purulent and presented a copious sediment of pus which contained a few colon bacilli.

Two days after the infection the fever had subsided the milk was yellow, thick and purulent and contained a rich purulent sediment which showed a few colon bacilli. Other conditions were the same.

Three days after the infection the swelling of the quarter had subsided. The quantity of the milk, however, remained small. The milk showed a normal appearance but was yellow and somewhat flocculent and contained a slight sediment consisting of yellow fibrinous pus containing a few streptococci. Upon cultivation of the sediment by plating a large number of streptococci but no colon bacilli were recovered.

Four days after the infection the milk was decreasingly less. The milk was of normal appearance but contained a smaller quantity of sediment consisting of yellow fibrinous pus showing few streptococci. It was otherwise as before.

Sixteen days after the infection this condition was unchanged. The animal was then slaughtered.

Autopsy.—The infected quarter was slightly swollen and its consistency a little firm. The incision was moist with milk. The lower part of the cut surface even, smooth, grayish-white and firm. The lobuli showed no milk points here. Upon pressure yellow fibrin cylinders were expressed (chronic streptomycosis).

The virulence of the colon bacillus in this case was somewhat great; the udder had shown a tendency to go

dry and in consequence of the chronic streptococcic infection presented less favorable conditions for the infection.

Consequently, I obtained a severe udder-infection with fever which was quickly eliminated by a catarrhal inflammation in three days in consequence of the conditions already named. The chronic streptococcic infection was at first concealed by this secondary infection but later again appeared and then became more severe.

The similarity to the experiment with staphylococci is apparent.

Udder-infection

The colon bacillus causes milk-infection and acute stroma-infection. These infections manifest a great similarity to those produced by staphylococci.

milk-infection [*Parenchymatous Mastitis*]

This is the most important. It is always primary and is caused by infection through the teat canal, seldom through infection of a fresh wound.

The local condition is usually confined to one quarter but can involve more quarters by infection (for example, during milking). The primary lesion varies with the virulence of the infectious organism and the condition of the udder. A superficial or moderately deep necrosis is the most common. A diffuse necrosis of the udder seldom occurs.

The quantity and quality of the inflammation varies with that of the primary lesion. The inflammation can be of a catarrhal or croupous nature. The infection shows no tendency toward chronicity.

Secondary infections may occur with strepto- and staphylomycosis, as well as with pyobacillosis and tuberculosis.

In this case the staphylomycosis can conceal the colibacillosis while the others as a rule may become masked by

the vigorous colibacillosis; but later, when the colon bacillus is eliminated, these chronic infectious conditions, streptomycosis, pyobacillosis, and tuberculosis, may proceed.

A saprophytic invasion of an original colibacillosis is infrequent.

General Conditions.—A secondary blood infection is seldom produced.

Intoxication varies with the virulence of the organism and the condition of the udder. Soon after calving the infection can cause a high-grade, probably fatal, septic intoxication accompanied by severe symptoms of fever.

Termination.—The simple colibacillosis is seldom fatal. The infection is eliminated by a more or less severe inflammation which lasts from three days to about three weeks. The subsequent sclerosis is usually of a low grade.

Clinical Symptoms.—The condition is always acute. The infected quarter is more or less severely swollen and hard. At first it is painful but later not sensitive.

The quantity of milk is diminished. The milk either forms a grayish-white, turbid serous fluid (primary lesion) or yellow, thick purulent fluid (inflammation which in severe primary lesions contains yellowish clumps of croupous membranes).

Upon microscopic examination of the sediment, Gram negative colon bacilli are seen as single rods or diplobacilli. They are somewhat large and thick and show rounded ends. In the stage of the primary lesion they are somewhat numerous, but in the stage of inflammation during the emigration of leucocytes their number is smaller.

The condition is accompanied with fever which may be very high. With the beginning of inflammation fever is reduced.

Prognosis.—In these cases the prognosis is always good, although in cows which have recently freshened the prognosis is less favorable.

Unfavorable symptoms are high fever and repressed leucocytic emigration, checked inflammation.

TREATMENT.—The condition is treated in the same manner as streptomycosis (see streptomycosis).

STROMA-INFECTION [*Interstitial Mastitis*]

The stroma-infection occurs only as a wound infection which is a complication of an udder-wound. It is an extremely harmless condition and usually presents a superficial wound necrosis with subsequent suppuration.

Secondary infection with strepto- and staphylomycosis is common and gangrene can also occur.

These wound infections, like the previous wound infections, are treated following the usual surgical methods.

SUMMARY

I. Infectious organism: the colon bacillus.
   - Characteristics.
   - Occurrence.
   - Morphology.
   - Cultivation.
   - Resistancy.
   - Pathogenicity.

II. Udder-infection. Udder colibacillosis.
   - A. Milk-infection [parenchymatous mastitis].
     - Local conditions.
     - General conditions.
     - Termination.
     - Clinical symptoms.
     - Prognosis.
     - Treatment.
   - B. Stroma-infection [interstitial mastitis].
     - Wound-infection.

CHAPTER IX

UDDER-PYOBACILLOSIS

Infectious Organism

The infectious organism is the pyogenes bacillus, bacillus pyogenes.

Characteristics.—The bacillus pyogenes is a very small thin bacillus. It stains slowly and faintly with the common anilin dyes but stains well after the method of Gram. Its growth on the usual culture media is thin, especially the surface growths. It liquefies coagulated serum and gelatin after a somewhat longer time. It coagulates milk. The organism is found in Germany, Holland, Denmark and Sweden. This indicates that it is of wider distribution, although it has not been reported since it was briefly studied by Grips, Glage and Nieberles. It can be found here and there in the various districts of Sweden.

It appears to be present frequently in the digestive tract of cattle since it is often found in diseased nodules produced by foreign bodies in the reticulum. One may therefore assume that it is to be found in the dirt and dust of our cow stables.

Morphology.—The bacillus pyogenes is a small and very fine rod. Its size varies somewhat. It is 1 to 3 microns long and 0.2 to 0.3 micron thick. As a rule, the bacillus is 2.5 microns long and 0.2 micron thick. In dimensions it most closely resembles the erysipelas bacillus and also the tubercle bacillus, except that the latter is a little larger.

The bacillus pyogenes is usually rod-shaped. Only in cultures, especially on coagulated serum, does the organism assume an oval or nearly round shape in consequence of rapid multiplication.

This organism usually appears singly in smear prepara-

tions. It seldom presents chains, which are short and composed of one or two, frequently three, individuals. The bacilli often lie parallel to each other or slightly converging so that groups may be formed (Fig. 19).

It is stained with difficulty by the common anilin dyes, but stains well after the method of Gram, but even here one must not too strongly decolorize it with alcohol. They are less Gram positive than the streptococci, but more so than the malignant œdema group.

Cultivation.—The bacillus pyogenes grows somewhat slowly at body temperature (38° C.) and very slowly and weakly at 20° C. It does not appear to grow at room temperature (15° C.). It grows aërobically and anaërobically, but shows more of an anaërobic character. In order to isolate it a pure culture medium containing serum is necessary, but after it has been isolated it can be grown on the common media without serum.

Fig. 19.—Pyogenes bacilli in milk sediment. Gram. (× 550).

In serum-gelatin-agar plates at 38° C. the growth is at first visible after from two to four days. The deep colonies in the first plate then appear as sparse small whitish-gray scarcely visible points the size of the hole made by a pin prick.

Under the microscope the colonies appear as very small bright yellow round or rounded structureless vesicles with even margins. The larger colonies appear to be produced by the grouping of a greater or lesser number of these vesicles into a composite mass like a raspberry and therefore present an irregular shape and a crenated margin. The smaller colonies are probably produced by one bacterium, " primary or single colonies," and the larger ones by coalescence of the primary colonies, " complex colonies " (Fig. 20).

As a rule one searches vainly for surface colonies and if he finds one it is extremely thin and consists of a scarcely visible whitish-gray round layer about 2 mm. in diameter.

In stab cultures in gelatin-agar and agar at 38° C. the bacillus pyogenes produces a growth in the line of the stab after two to five days which resembles a thread. Usually no growth occurs on the surface, but when present appears as a fine gray point over the opening in the stab puncture (Fig. 21). In gelatin stab cultures the growth is slow and scanty. It also forms a thread-like growth along the line of puncture in the media which shows infundibuliform liquefaction along the stab after a week, following which the bacteria form a small deposit on the bottom made by the firm media (Fig. 22).

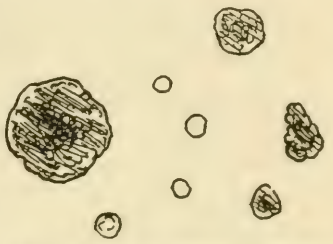

Fig. 20.—Deep colonies of pyogenes bacilli. Serum-gelatin-agar plates. T. 38°, 2 days (× 30).

On streak cultures on coagulated serum at 38° C. it forms a fine scarcely visible gray thread along the streak in about four days. After six days on the media liquefaction occurs forming a moist glistening groove along the streak.

Fig. 21. Fig. 22.

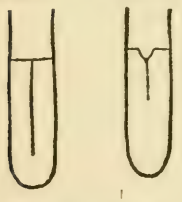

Fig. 21.—Stab culture of pyogenes bacilli in serum-gelatin-agar. T. 38° C., 4 days.

Fig. 22.—Stab culture of pyogenes bacilli in gelatin. T. 20° C., 12 days.

In bouillon the bacillus grows in from two to four days at 38° C. without turbidity of the media, but shows small fine gray granules which sink to the bottom of the tube, forming a granular deposit. Upon shaking the tube these deposits cause the media to become diffusely turbid.

The bacillus pyogenes coagulates milk at 38° C. in two to three days and the coagulum thus formed later liquefies with the formation of whey.

In dextrose media the bacillus pyogenes produces acid but no gas. The media can then contain 1.3 per cent. normal acid. In maltose, lactose, and saccharose media it forms neither acid nor gas.

Resistancy.—The bacillus pyogenes is very sensitive. Usually daylight can kill it in culture in a few days. It can be kept for two months in cultures not exposed to the light and sealed to preserve moisture.

Pathogenicity.—B. pyogenes is a typical pus producer and induces its effect slowly. Therefore it does not cause an acute but a chronic infection. It causes suppurative foci at the point of inoculation. The animals never die of bacteræmia.

Mice manifest a subacute infection usually with no alterations. One mouse, however, showed a chronic suppurative wound at the point of infection fourteen days after inoculation.

Dogs frequently but not always present a subacute infection after about fourteen days and show abscesses containing thick yellow pus and numerous pyogenes bacilli. Upon intraperitoneal inoculation similar abscesses are produced in the omentum and mesentery. Otherwise the health of the animal does not appear to be disturbed.

Two *guinea-pigs* infected subcutaneously manifested no alterations after two months.

Cows.—1. About six months after calving a cow was injected in one teat with 12 c.c. of a three-day-old bouillon culture of bacillus pyogenes which had been isolated from a case of calf pneumonia. This bacillus had been grown on culture media for a period of three weeks. The dog used as a control developed a distinct abscess at the point of injection fourteen days after inoculation.

Examination of the quarter and its milk before the infection revealed no pathological alterations.

The day after infection the body temperature was nor-

mal. The quarter was not changed. The quantity of milk was not diminished. The milk had a normal appearance but presented a small quantity of sediment composed of cells. The cells contained one single bacillus pyogenes.

On the third day after infection the quarter was unchanged. The milk was unaltered.

The same conditions obtained on the following day.

Nine days after the infection the part near and behind the teat (this was a hind quarter that was infected) was swollen and presented a distinctly circumscribed round, hard, sensitive swelling the size of a hen's egg. The milk was not changed.

This condition was the same on the following day.

Seventeen days after the infection the skin of this part of the udder was œdematously swollen, firm and immovable and adhered to the swelling of the udder. The milk was yellow and flocculent and contained a moderate sediment which consisted of pus containing groups of bacillus pyogenes.

Twenty-one days after the infection this condition remained the same. The animal was then slaughtered.

Autopsy.—The infected quarter was swollen and firm around the base of the teat. The incision was purulent. The lower part of the cut surface even, gray and firm. The lobuli showed no milk points, but instead small pus points up to the size of a pinhead. Upon pressure yellow plugs of pus were expressed. Externally at the upper part of and behind the base of the teat there was a cavity the size of a walnut with thick connective-tissue wall and irregular lumpy interior. It contained a greenish-yellow, thick pus which showed large numbers of small fine Gram positive pyogenes bacilli.

2. Three weeks after parturition another milch cow was injected in one teat with 10 c.c. of a mixture consisting of equal parts of bouillon culture of streptococci and

bouillon culture of bacillus pyogenes. The streptococcus and also the bacillus pyogenes were isolated from a case of mastitis. The streptococcus killed two control mice, the first in one and the second in two days. The virulence of the bacillus pyogenes was not controlled.

Examination of the infected quarter and its milk before infection revealed no pathological alterations.

On the day after infection the body temperature was 40.5° C. (ca. 105° F.). The quarter was severely swollen and painful, the quantity of milk was decreased and the milk was yellowish-white and thick and showed sediment consisting of pus which contained a great number of streptococci but no pyogenes bacilli.

Two days after the infection the body temperature was 39.8° C. (ca. 103.5° F.). The milk was yellow and purulent and contained shreds of croupous membrane showing a few streptococci. It was otherwise as before.

Three days after the infection the body temperature was 39.3° C. (102.5° F.). It was otherwise the same as before.

Four days after the infection the body temperature was normal. The swelling of the quarter was somewhat reduced. It was otherwise the same as before.

During the following day this condition remained the same.

Six days after the infection the quarter showed firm nodular swellings here and there. The milk was yellowish-gray, purulent and contained croupous membranes which showed a few streptococci and here and there in the cells small groups of pyogenes bacilli.

During the following days this condition remained unchanged.

Twenty days after the infection the quarter was in the same condition. The milk was yellow and pus-like and contained free croupous membranes showing few streptococci

but many pyogenes bacilli which were for the most part within the cells. The animal was now slaughtered.

Autopsy.—The infected quarter was moderately swollen, irregular, nodular and of firm consistency. The incision was smeared with a thick yellowish-green pus. The cut surface was even, yellowish-gray and firm. The lobuli showed no milk points, but showed pus points distinctly. Upon pressure yellow pus cylinders were expressed. Externally seven suppurative foci were observed which ranged in size from a hazelnut to a walnut. They contained yellowish-green cheesy pus and were surrounded by a white thick, firm connective-tissue capsule. Numerous milk-ducts formed thick firm cords which on cross section presented yellowish-green pus centres and were surrounded by a white, thick, firm layer of connective tissue (fistula formation). The yellowish-green pus contained a great number of small thin Gram positive pyogenes bacilli.

Both of these experiments are good examples of infection through transportation of the infectious organism by the leucocytes. The infected milk is quickly freed from the organisms by resorption, that is, in these cases the leucocytes pick up the organisms and carry them away.

In the lymph-vessels a few of these phagocytes are destroyed by the living bacteria which they contain and these liberated organisms induce a stroma-infection which finally terminates as a secondary milk-infection by perforative ulceration of the milk-ducts.

Bacillus pyogenes so restrains itself that it simulates a saprophyte and leucocytes may pick it up and re-enter the vessels, where later it manifests its parasitic nature and then destroys these cells. It is therefore insidious in its action. It might be classed as a cell parasite instead of a fluid parasite because of this action toward cells.

In the first experiment the action of the bacilli was slower and less widespread, because the alactiferous (dry)

udder afforded less favorable conditions for development. In the last case the bacterial action was quicker and more widely distributed, partly because the udder was rich in fluid which afforded a better nutrient medium for the micro-organisms and partly because of the synchronous strepto-coccic milk-infection, circumstances, so to speak, which placed the tissue between two fires.

Udder-infection

Bacillus pyogenes always induces a stroma-infection first, and this can later develop into a secondary milk-infection by perforative ulceration of the milk-ducts. Therefore the stroma-infection is the most important, and milk-infection is only a result of it.

stroma-infection [*Interstitial Mastitis*]

The stroma-infection manifests a typical progressive character.

Ports of Infection.—Infection may gain entrance through the teat canal, wounds, or through the blood stream. Infection by way of the blood follows pyobacil-losis of the lungs with penetration of a pulmonary vein by a necrotic focus. Pyobacillosis of the lungs is not alto-gether infrequent. It appears to be present in calves prin-cipally and may cause udder conditions in yearling heifers. The condition can be produced through an infected wound and is not uncommon.

Infection through the teat canal is the most common, in my opinion, since the condition appears to be primary in the udder without presenting any wound of the udder. If the bacillus is brought to the mouth of the teat canal it can enter the milk in that canal and grow in it, and then be taken up by the leucocytes. In other cases a cow may lie down and the infection be introduced on a straw which may be forced into the teat canal.

Local Conditions.—The lesions produced in the udder occur in proportion to the number of organisms which gain entrance. The bacilli are taken up by the leucocytes in the capillary blood, in wounds, or in the milk and deposited in the stroma of the udder, where the leucocytes are finally necrotized by the parasites. In many cases perhaps the opposite condition takes place, that is, the bacilli are destroyed and the infection prevented. The infection can occur, however, by entrance of great numbers of bacteria, that is, many bacteria; but still more leucocytes are destroyed and the infectious organisms become superior to the leucocytes.

An inflammatory zone forms around these necrotic cells (primary lesion) and outside of this zone of leucocytes a capsule of connective tissue develops. Secondary nodules are formed during the removal of the infectious organisms by the leucocytes. All of these foci gradually enlarge with subsequent atrophy of the tubules and finally ulceration into the tubules or outward through the wound, if one be present, or through the skin.

Depending upon the point of entrance of the infection the following forms may be distinguished, *i.e.,* (1) primary udder-pyobacillosis; (*a*) udder-pyobacillosis through teat infection; (*b*) udder-pyobacillosis through wound-infection. (2) Secondary or embolic udder-pyobacillosis.

Primary udder-pyobacillosis usually occurs without pyobacillosis of the lungs. It may, however, be present along with the lung condition, in which case the latter may be either primary or secondary (caused by blood-infection induced by udder conditions).

Udder-pyobacillosis through teat infection usually affects a single quarter, but can then reach other quarters through infection, *i.e.,* during milking. It starts around the base of the teats and then spreads further sideways and upward.

Udder-pyobacillosis through wound-infection is distinguished by its localization in the neighborhood of the wound and by a comparatively uniform distribution of the condition from this point inward. Ulceration in the wound is great and tends toward fistula formation.

Secondary or embolic udder-pyobacillosis is distinguished by a primary condition of the lungs and by its position, which usually is in several quarters in the beginning, seldom in one quarter alone. Should one quarter alone be affected it is most likely to be a hind quarter. It is further distinguished by large and somewhat uniform distribution in the affected quarters in the beginning. (The condition is distributed in the lower parts in most instances, however.)

In the beginning these forms may be sharply separated. Through development of the conditions they may become somewhat similar. Thus, for example, primary udder-pyobacillosis through wound-infection showing secondary ulceration through the skin with subsequent fistula formation can become very difficult to distinguish from the other forms.

By ulceration through the milk-ducts pus containing infectious organisms is emptied into the milk. This pus can: (1) Be withdrawn with the milk without producing any changes in the udder; (2) the infectious organisms can be picked up in the milk by fresh leucocytes and be again carried into the connective tissue, through which the infection is spread further, and (3) the thick pus can remain in the milk-ducts for a shorter or longer time, through which the infection, as a secondary milk-infection, can cause more or less necrosis of the underlying tissue and thus produce a croupous inflammation. Upon desquamation of this croupous membrane a wound is presented which likewise becomes infected.

Secondary infections are common and appear to aggra-

vate the condition greatly and to favor the primary infection. They may be divided into milk- and stroma-infections.

Secondary Milk-infection [*Secondary Parenchymatous Mastitis*].—These infections may be strepto- or staphylomycosis as well as colibacillosis. The most common, however, is the streptomycosis. They occur one at a time as a rule, but several secondary infections may be present at the same time.

These secondary infections, especially staphylomycosis, may raise the condition to an acute process and thus induce diffuse necrosis of the quarter and cause the death of the animal. In the earlier stages of pyobacillosis they not infrequently mask the primary condition.

In developing, pyobacillosis (with secondary milk-infection) favors the formation of croupous exudate which begins to form in considerable quantities in the secondary infection. This exudate can remain situated in a teat canal and in this way form a good bridge for the infectious organisms.

Secondary Stroma-infection.—These secondary stroma-infections may be of two different kinds, acute and chronic.

Acute secondary stroma-infection occurs only as a secondary infection to wounds. It presumes, consequently, a primary or a secondary wound produced by ulceration. In these wounds secondary infection is the rule. These secondary infections may be strepto- or staphylomycosis or colibacillosis. They may occur one at a time or several secondary infections may be present together.

The single chronic secondary infection which appears to occur is tuberculosis. It is infrequent. Pyobacillosis and its secondary infection, which it somewhat resembles in nature, work together for the destruction of the udder. A

secondary infection with actinomycosis is possible but occurs infrequently.

Saprophytic invasion is common and appears to favor the infection. It causes milk- or (and) wound-gangrene.

Milk-gangrene is very common. The croupous exudate in extensive pyobacillosis with secondary milk-infection predisposes toward this complication. This exudate as before mentioned can remain located in the teat canal and constitute a good bridgework. Secondary milk-infection also favors the beginning of this complication to a great extent.

Wound-gangrene may develop in a primary or secondary wound. It is very common and we may say it is the rule if wounds are not treated.

General Conditions.—*Blood-infection.*—A necrotic focus may contain a vein which becomes open by perforative ulceration and admits the bacteria which caused the necrosis. These organisms are carried by the venous system to the lungs. From the lungs the bacteria may enter the pulmonary veins in a similar manner and be carried by the arterial blood stream to one or several other organs of the body, inducing secondary metastatic foci.

Intoxication.—Pyobacillosis causes a low-grade chronic intoxication, which, like the lesion, has a progressive character. It continues to a chronic progressive emaciation and anæmia with thickening and finally a leathery skin and roughened hair coat and finally to fever and death of the animal.

Complications with acute secondary infections and [or] with gangrene often produce high and continuous fever and may terminate more or less quickly in death of the animal.

Termination.—The primary condition seldom causes death. It advances by a chronic protracted course, extending over months and years to progressive destruction of

the infected quarter and not infrequently presents similar secondary lesions in other organs. It predisposes to the secondary infections and gangrene, which conditions, especially together, terminate in death.

In one case the infection confined itself to the cisterns and milk-ducts, to the exclusion of the udder which was nearly free from the infection. This form of the infection induces destruction of the mucous membrane of the milk-ducts with subsequent healing and connective-tissue formation, which closes the lumen of the ducts, with the result that the cisterns present a white vault-like connective-tissue dome without any openings for the milk-ducts. In consequence of these alterations the udder shows milk stasis with distended lobuli and tubuli together with a firm condition due to sclerotic connective-tissue proliferation.

Clinical Symptoms.—The condition is chronic. One or more quarters are affected. The diseased quarters are nodular and swollen in their lower extremities. These nodular foci are hard and painful or soft and then fluctuating. Sometimes one sees a chronic wound or fistula in these foci which presents a stinking secretion indicative of gangrene. The nodular foci develop slowly.

At first the quantity of milk is only slightly lessened, but later very much diminished. In quarters which have not undergone very marked changes the milk is of almost normal appearance. Later when they have become more extensively altered the milk shows clumps of pus, since the milk is mixed with pus, and finally it is yellow, thick and purulent and contains free fibrin shreds. In secondary infections the milk can become grayish-red, and in gangrene it has a foul odor. The odor is often characteristic and it is specific for pyobacillosis. It resembles rotten cabbage.

Upon microscopical examination of the sediment one

usually perceives a great number of Gram positive pyogenes bacilli outside of the cells.

When uncomplicated the condition is free from fever, but passes on to chronic emaciation with leather-like thickening of the skin and roughened hair coat. In complications with secondary infections or (and) with gangrene fever develops which may be high and continuous.

The above mentioned case with localization of the infection principally in the cisterns and milk-ducts presents a somewhat diverging picture. The affected quarter is uniformly swollen and hard. The milk is perhaps of normal appearance or may show fibrin flakes. The quantity of milk decreases somewhat slowly and tends to terminate after one to two months, or longer, by complete suppression of milk secretion.

Prognosis is always unfavorable. Healing of this udder condition is possible but very infrequent. A progressive destruction of the organ is the rule. The affection predisposes to fatal complications with secondary infections and gangrene. With manifestation of such conditions, high fever and red-stained or stinking milk, the prognosis is unfavorable.

Treatment.—Prophylaxis is the most important. If a stable of cows contains one case of udder-pyobacillosis one should slaughter the affected cow in order to prevent further spreading of the infection. If the owner will not consent to this the animal must be strictly isolated. She is milked last and the infected milk must be so treated with disinfectants that the living infectious organisms cannot be further distributed. For prevention of a possible transmission of the infection by means of flies, the teats and wounds, if wounds be present, of the affected cow may be smeared with creolin-petrolatum, which the flies avoid because of its odor.

By *actual treatment* one endeavors, when possible, to

bring about a sclerosis and atrophy of the affected quarter. In this way the progressive destruction of the udder is prevented, healing is possible and at the same time the udder becomes less susceptible to secondary infections.

Deep point firing or cautery in a completely infected quarter is indicated since the treatment greatly favors sclerosis and contraction.

If fluctuation is observed the abscess is opened with a scalpel, the pus emptied, and the inside of the cavity thoroughly cauterized with a cautery iron.

If a secondary milk-infection [parenchymatous mastitis] is complicated with it, this is additionally treated with injections of boric acid solution and inflation with air. If indications point toward death of the animal one may possibly attempt to prevent it by extirpation of the diseased half of the udder.

In gangrene accompanied by high fever one can amputate the teat in order to induce free drainage of the secretion.

In fever a febrifuge like camphor is indicated.

Summary

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   Resistancy.
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II. Udder-infection. Udder-pyobacillosis.
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   Forms:
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Secondary infections.

1. Milk-infection [parenchymatous mastitis].
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1. Milk-gangrene.
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General conditions.

Blood-infection.

Intoxication.

Termination.

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CHAPTER X

UDDER-TUBERCULOSIS

Infectious Organism

The infectious organism is the tubercle bacillus, bacillus tuberculosis. This bacillus is well known and therefore I shall omit its description.

I will mention, however, that this organism differs from the preceding in that it is a strict parasite and therefore its occurrence is confined to the animal host. It has a very great geographical distribution nevertheless. In Sweden it is common in the southern and middle parts, but is less frequently found in the north.

When excreted from the body of the host in pathological discharges it cannot multiply under common conditions as a strict parasite. Although it does not form spores it does, however, obstinately resist harmful external influences as drying or mixing with other bacteria like those of putrefaction, and it may live for a month in dark places and retain its virulence.

Transmission of the infectious organism does not demand direct contact of one animal with another, but can be brought about by objects like straw which have become contaminated by pathologic discharges containing the tubercle bacilli. Indirect transmission, indeed, seems to be the most common.

I have never inoculated a cow's udder with tubercle bacilli, but with an experiment one could depend upon the same conditions as those obtained in the experiment with bacillus pyogenes, *i.e.*, that the bacilli are taken up by the leucocytes through which the milk becomes free from the bacilli, and after one week, fourteen days or longer after

the infection, bacilli again appear in the milk. That is to say, the tubercle bacillus like the bacillus pyogenes is a typical cell parasite.

Udder-infection

Udder-infection is not altogether infrequent. Figures taken from statistics on tuberculosis in the Malmoer slaughter houses for 1905 showed ca. 3.5 per cent. of tuberculous cows to be affected with udder-tuberculosis.

The tubercle bacillus always presents a typical stroma-infection which can, however, develop into a secondary milk-infection by ulceration of the milk-canals. Stroma-infection is thus of the greatest importance.

stroma-infection [*Interstitial Mastitis*]

This infection has a typical chronic and progressive character.

Ports of Infection.—The infection can enter through the teat canals, through wounds or through the blood.

The infection through the blood is the most common. It is one which follows tuberculosis of the lungs with blood-infection following ulceration of veins.

If a blood-infection is produced by any other organ following ulceration of a vein or through transportation by lymph through the thoracic duct, the infectious organisms as a rule are picked up in the lung capillaries by the leucocytes and carried into the lung tissue, by which process the blood is freed from the organisms. It is, however, possible that one or more bacilli may not be carried into the tissue of the lung, but pass through the lung and through the pulmonary veins, ultimately reaching the blood of the aorta and thus perhaps be carried to the udder. Thus it is possible, although not frequent, that infection of the blood of the udder is caused by tuberculosis in organs other than

the lungs. In such cases the foci in the lungs and in the udder are of the same age, the former more numerous than the latter, however.

As a rule, infection of the blood in the udder is caused by tuberculosis in the lungs. Only exceptionally is udder tuberculosis caused by tuberculosis in other organs, in which the udder- and lung-infection occur at the same time.

Infection through wounds is infrequent. I have only seen one case of this kind. The wound becomes infected by contamination with tuberculous products, such as coughed-up tuberculous mucus. The contamination can be transmitted by straw or the fingers of the attendants.

Infection through the teat canal is less frequent than by way of the blood stream, but more common than the wound infection. The latter comparison is only natural, since the teat canal is always present while udder wounds are somewhat infrequent.

It is unreasonable to think that tubercle bacilli which grow so very slowly can develop in the milk of the teat canal, but one must consider that the infectious organism is mechanically forced into the teat canal on a straw, for example, which has been contaminated by coughed-up tuberculous mucus.

If the udder is engorged with milk the distended teats will be as firm as wood and the teat canals will be somewhat widened as a result of this distention. Thus a hard straw may be forced into the teat canal by movements of the animal while lying down.

Another possibility of infection is by the leucocytes picking up the infectious organisms and carrying them into the udder. This, however, always necessitates contamination of the point of the teat by tuberculous products either directly or indirectly by straw or by the hand of the milker.

Local Condition.—The lesions are in proportion to the infectious material taken into the udder. The bacilli

are taken up by the leucocytes in the blood, in wounds or in the milk and carried into the lymph spaces of the stroma, where the leucocytes are finally killed by the bacilli contained within them.

Primary Lesion and Inflammation.—Around these necrotic cells which constitute the primary lesion, a number of cells are attracted, in the process of inflammation, and around these a layer of new connective tissue forms. This structure is the primary tubercle.

By progressive necrosis a figure called a giant cell is formed in the centre. It is this round or oval necrotic focus which presents a zone of cell nuclei upon its external border which are not destroyed by the organism within its structure. The necrotic focus thus resembles a large cell with many nuclei, giving rise to the name giant cell (Fig. 23). The organisms multiply within the tubercle very slowly.

Fig. 23.—Tuberculosis of the udder. Stroma-infection [interstitial mastitis]. Primary cell necrosis and two giant cells in the intralobular connective tissue with corresponding inflammatory zones of lymphocytes and wall of connective tissue. Tubercle bacilli were demonstrated in the area of necrosis with bacterial stains.

[H. F. Smyth, in his work on tissue culture, has shown that tissue inoculated with tubercle bacilli and grown *in vitro* will form giant cells by fusion of epithelioidal cells. The giant cells are forty or fifty times larger than epithelioidal cells. Giant cells may be seen anywhere in a tubercle, which usually presents a centre of cheesy necrosis surrounded by a zone of epithelioidal cells, outside of which is a zone of lymphocytes, which is later surrounded by a layer of connective tissue.]

Distribution.—The infection manifests a great tendency to spread. A few bacilli in the primary tubercle may be picked up by leucocytes and be carried further into the stroma in the direction of the lymph stream where these leucocytes may be destroyed by the organisms and daughter

tubercles be produced. This is local metastasis or extension by leucocytes.

In this manner the organisms can gain entrance to the lymph-vessels and be carried to the supramammary lymph-glands, where the degenerated leucocytes are retained and a tubercle is produced. Thus tuberculosis of the lymph-glands depends upon infection of the lymph and the process is therefore called lymphogenous metastasis.

Tuberculosis in the lymph-glands following such lymphatic metastasis is more recent than the tuberculosis of the udder. Thus we may have distinct caseation in the udder, but subsequent caseation in the lymph-glands may frequently be so insignificant that it is not visible to the naked eye.

From these lymph-glands leucocytes may carry the organisms through the lymph to the lumbar-glands and from here by way of the thoracic duct to the blood stream, by which it may finally be widely distributed. Thus lymphogenous metastasis may be transformed into hæmatogenous metastasis.

Besides these forms of metastasis it may also be transported through milk [galactogenous metastasis] and blood, which will be discussed in another chapter.

Development of the Tubercle.—The primary tubercle manifests slow development. Central necrosis progresses gradually. During the lactation period, however, the tubercle develops somewhat rapidly in the succulent stroma while it develops very slowly in the alactiferous udder.

Adjacent primary tubercles may coalesce by extension. Their connective-tissue walls and cellular zones join first and finally the necrotic centres coalesce and form a large area of necrosis surrounded by a wider zone of lymphocytes and connective tissue. By coalescence of a large number

of primary tubercles a very large necrotic nodule may be formed.

Secondary Alterations of the Necrotic Mass.—This necrotic mass, which is yellowish-white in color and is firm and caseous through coagulation, can undergo certain changes.

It can become swollen and softened and be transformed into a pulplike mass by the imbibition of lymph, made possible during the lactation period by increased blood- and lymph-supply to the organ.

Under reverse conditions, that is scarcity of lymph, as in the alactiferous udder, they can become dry, firm and finally by resorption of liquid and infiltration of calcium salts become partially calcified. The calcification begins in the centre of the necrosis and progresses toward the periphery. Thus, through resorption of liquid and calcification, the area of necrosis forms a concrement which is situated in the tissue of the stroma. This concrement may be called a tissue concrement to distinguish it from the liquid concrements which are formed in milk, urine, bile and other fluids.

In the softening of an area of necrosis during a reverse process, sandlike granules or sediment may form in the pulplike necrotic mass, due to infiltration of lime.

Disturbance of Circulation by Tubercles.—Since tubercles are situated in the lymph-spaces of the connective tissue, they finally obstruct the circulation of lymph if they occur in great numbers. The lymph stagnates in the lymph-vessels in the most posterior portions of the hind quarters of the udder. In this manner œdema is produced in the quarter affected, likewise in the entire half of the udder, following tuberculosis of the supramammary lymph-glands. In this condition these glands and finally the lumbar-glands become œdematous.

Disturbance of Milk Excretion by Tubercles.—The tubercles usually form in the lower parts of the udder and as they develop exert pressure upon the walls of the adjacent milk-ducts, inducing their collapse, obstruction of milk flow and finally milk stasis [galactostasis] in the superior parts of the udder. The tubules and finally the lobules are distended by the stagnated milk. Milk concretions may form in the dilated closed tubules through resorption of water and precipitation of the less soluble matter. These are exceptionally small granules which are only visible with the microscope. They resemble granules of starch in appearance and therefore are called corpora amylacea.

Milk stasis is seldom severe enough to induce pressure atrophy, and finally rupture of the walls between tubules to the formation of one large single tubule or milk cyst [galactocele] by coalescence of two or more tubules.

Ulceration of Milk-ducts.—Tubercles may destroy the walls and epithelium of the milk-ducts. Direct contact with the milk increases the liquid content of the tubercle and in this way the necrotic area undergoes softening and parts of it are finally emptied into the milk.

In this way the tubercle bacilli, like the pyogenes bacilli: (1) may be excreted with the milk; (2) be transported to other parts of the same quarter by leucocytes, or (3) cause a secondary milk-infection by remaining in the milk-ducts with the thick pulp-like mass of softened necrotic material.

The retained infectious organisms gradually induce necrosis of the mucous membranes, which desquamate and, like croupous membranes, are excreted with the milk. The wound produced by desquamation of these necrotic cells can become infected and thus the condition proceeds.

Ulceration of a Blood-vessel Wall.—A tubercle can destroy a vein wall in a similar manner. The altered

vein wall usually induces a thrombotic process, however, which, as a rule, prevents infection of the blood. Ulceration finally occurs, however, in the larger veins which are not occluded by the thrombus. In this case the thrombus is broken up by the softened tuberculous necrosis which is ultimately emptied into the blood stream. In this way a secondary blood-infection is produced which metastasizes to the lungs.

Blood-infection in consequence of this thrombotic process always occurs later and on a smaller scale than the milk-infection described.

Great numbers of such venous thrombi cause venous stasis with subsequent transudation by which the œdema, which has already been caused by lymph stasis, is increased.

Arterial walls may be destroyed in the same manner, following which the condition may be more widely distributed by infection of the arterial blood.

Tuberculous Wounds.—If the tuberculous lesions lie near the skin, they may ultimately cause its necrosis and the softened tuberculous necrotic substance may be discharged from the wound so formed. This is somewhat rare.

If the tuberculous processes lie adjacent to a primary wound or a secondary wound produced by ulceration, the tuberculous processes may ulcerate into these wounds.

Types.—Depending upon the ports of infection, as in pyobacillosis, the following forms of udder-tuberculosis are distinguished: (1) primary udder-tuberculosis; (*a*) by infection through the teat canal; (*b*) by infection through wounds; (2) secondary or embolic udder-tuberculosis.

These types have the same differential characteristics in general as the forms of pyobacillosis described.

Primary udder-tuberculosis following teat infection is comparatively rare. Of 137 cows which had udder-tuberculosis, slaughtered at the expense of the state, nine, or

6.6 per cent., did not show tuberculosis of the lungs, according to the post-mortem protocol. These were cases of primary udder-tuberculosis which were obviously caused by infection through the teat canal, since the udders presented no wounds.

Since this type of primary udder-tuberculosis can be present along with tuberculosis of the lungs, this figure is somewhat too small. It should probably not be too much increased, however. If we estimate, that ca. 10 per cent. of all cases of udder-tuberculosis are primary and caused by infection through the teat canal I believe that we shall be more nearly correct.

It is absolutely impossible to distinguish primary from embolic tuberculosis of the udder by post-mortem examination. Primary tuberculosis following infection through the teat canal presented no specific post-mortem picture, but the lesions are the same as those of embolic udder tuberculosis.

Likewise, localization of lesions in one quarter is of no special significance, since embolic tuberculosis can begin in any quarter. Further, it appears that the condition can easily spread from one quarter to another by teat-canal infection. The infection can also be extended by the lymph stream, but only in the direction in which the lymph flows from the fore quarters toward the hind quarters.

Of the nine cases of primary udder-tuberculosis induced by teat-infection named, five cases showed only one quarter affected: the left posterior quarter in two cases; the left anterior quarter in one case; the right posterior quarter in two cases; three cases showed two quarters affected: both posterior quarters in two cases; the left posterior and right anterior quarter in one case; one case showed all four quarters affected.

Primary Udder-tuberculosis by Infection of Wounds.— This is exceptionally rare. I have only seen a single case

of this kind. According to reports, the udder of the cow had been hooked, inducing a wound. The wound gave rise to a fistula. A number of tubercules were produced in the walls of the fistula and adjacent udder-tissue which were very similar in character.

Secondary or Embolic Udder-tuberculosis.—This type, as before stated, is the most common and constitutes approximately 90 per cent. of all cases.

In 115 cases of udder-tuberculosis with synchronous lung-tuberculosis, that is in 115 cases which were exclusively of the embolic type of udder-tuberculosis, the condition is designated in the different quarters in the following manner:

A. In one quarter:

In left posterior quarter	28 udders, *i.e.*,	23 per cent. of cases
In left anterior quarter	5 udders, *i.e.*,	5 per cent. of cases
In right posterior quarter	16 udders, *i.e.*,	14 per cent. of cases
In right anterior quarter	5 udders, *i.e.*,	5 per cent. of cases
Summary	54 udders, *i.e.*,	47 per cent. of cases

B. In two quarters:

In both posterior quarters	13 udders, *i.e.*,	11 per cent. of cases
In left posterior and anterior quarters	6 udders, *i.e.*,	5 per cent. of cases
In right posterior and anterior quarters	13 udders, *i.e.*,	11 per cent. of cases
In left posterior and right anterior quarters	2 udders, *i.e.*,	2 per cent. of cases
In right posterior and left anterior quarters	4 udders, *i.e.*,	4 per cent. of cases
Summary	38 udders, *i.e.*,	33 per cent. of cases

C. In three quarters:
In both posterior quarters and right anterior quarter 5 udders, *i.e.*, 4 per cent. of cases
In both posterior quarters and left anterior quarter 7 udders, *i.e.*, 6 per cent. of cases
In both anterior quarters and in left posterior quarter ..... 1 udder, *i.e.*, 1 per cent. of cases
In both anterior quarters and in right posterior quarter ..... 2 udders, *i.e.*, 2 per cent. of cases

Summary .....15 udders, *i.e.*, 13 per cent. of cases

D. In all four quarters..... 8 udders, *i.e.*, 7 per cent. of cases

Totals .....115 udders

The quarter most frequently affected according to the following table.

In the above named 115 cases tuberculosis occurred:

A. In left posterior quarter...70 udders, *i.e.*, 61 per cent. of cases
In left anterior quarter...33 udders, *i.e.*, 29 per cent. of cases
In right posterior quarter.68 udders, *i.e.*, 59 per cent. of cases
In right anterior quarter..36 udders, *i.e.*, 31 per cent. of cases

B. In left half of the udder...81 udders, *i.e.*, 70 per cent. of cases
In right half of the udder..76 udders, *i.e.*, 66 per cent. of cases
Note:
Exclusively in left half of udder .....39 udders, *i.e.*, 34 per cent. of cases
Exclusively in right half of udder .....34 udders, *i.e.*, 30 per cent. of cases

C. In one or both posterior quarters .....105 udders, *i.e.*, 91 per cent. of cases
In one or both anterior quarters .....58 udders, *i.e.*, 50 per cent. of cases
Note:
Exclusively in posterior quarters .....57 udders, *i.e.*, 50 per cent. of cases
Exclusively in anterior quarters .....10 udders, *i.e.*, 9 per cent. of cases

The left half of the udder is somewhat more severely exposed to the infection than the right. The posterior quarters are much more frequently infected than the anterior quarters. The anterior quarters are seldom infected. Usually one or two quarters, not frequently more than that, are affected.

Embolic tuberculosis of the udder occurs in one of the posterior quarters as a rule, and in addition to it, in the anterior quarter on the same side, or the other posterior quarter or in both of these, *i.e.,* both posterior quarters together with one anterior quarter. Less frequently there are infected only one of the anterior quarters, or one posterior quarter and the anterior quarter on the opposite side, or all four quarters together.

Lung-tuberculosis in calves is somewhat infrequent, but is common in old cows. One may expect, therefore, that udder-tuberculosis will occur principally in old animals. Of 72 cases of udder-tuberculosis: 1 animal (1 per cent.) was 3 years old; 11 animals (15 per cent.) were 4 to 6 years old; 60 animals (84 per cent.) were 7 years old or older.

Secondary Infection by Teat-infection.—This is comparatively rare, but can occur nevertheless. A complication with chronic streptococcic infection appears to be the most common. Its effect is such that the milk becomes quickly altered and pus-like. This condition induces a severe sclerosis which is inhibitive to the extension of tubercles. In such an udder a few small tubercles are observed in the indurated parts, while the surrounding portions, which have not been affected by the streptococcic infection, present numerous large tubercles.

Acute milk-infection [parenchymatous mastitis], like acute streptomycosis, staphylomycosis and colibacillosis, may develop secondarily in chronic udder-tuberculosis and then conceal the primary chronic condition by its intensity. This complication appears to be quite rare.

Frequently secondary pyobacillosis develops wherein the organisms which produce an effect similar to that of the tubercle bacilli in general concur in the destruction of the udder.

I have never observed a secondary infection with actinomycosis.

Once I observed a secondary infection with an organism closely allied to the diphtheria bacillus (and b. pyonephritidis bovis). The bacillus was Gram positive, moderately large, and when grown on coagulated blood serum showed granular inside bodies capable of taking a deep stain. Cultures in serum-gelatin-agar plates, stab serum-gelatin-agar, coagulated blood-serum, and in milk and bouillon were like the diphtheria bacillus, yet no pellicle was formed on the surface of the bouillon. One cubic centimetre of an eight-day bouillon culture injected subcutaneously killed a guinea-pig by intoxication.

In addition to numerous small tuberculous foci near the base of the teats the udder presented five grayish-red firm necrotic foci ranging in size from a hazelnut to a walnut and surrounded by a connective-tissue capsule. Besides tubercle bacilli, these nodules showed a great number of the above-mentioned bacilli. I have never seen this condition occur as a single infection of the udder.

Secondary Infection Through Wounds.—These secondary infections occur from a primary wound or from a secondary wound produced by ulceration. Such wounds produced by tuberculous ulceration are rare, however.

These secondary infections may be streptomycosis, staphylomycosis, colibacillosis and pyobacillosis. They may occur separately or several together.

Staphylomycosis liquefies the necrotic area and consequently induces an infectious softening.

Saprophytic invasion can cause milk-gangrene and wound-gangrene.

Milk-gangrene does not appear to occur in simple udder-tuberculosis.

I have only seen a few cases of milk-gangrene in udder-tuberculosis and they were complicated with pyobacillosis.

Wound-gangrene presupposes a tuberculous wound. In this kind of wound gangrene is the rule. It develops into putrid liquefaction and softening of the necrotic tuberculous area.

General Conditions.—*Blood-infection.*—Tuberculosis of the udder can induce an infection of the blood-stream by lymphogenous transportation or by ulceration of a vein with subsequent metastasis to the lungs, from which point the condition can be more widely distributed.

Udder-tuberculosis, which is usually caused by lung-tuberculosis, can finally increase the severity of the condition in the lungs by metastasis.

Intoxication.—Like all tuberculosis of cows, udder-tuberculosis produces no distinct initial general symptoms.

The animal emaciates gradually and shows anæmia, decreased appetite and frequently leather-like thickening of the skin and roughened hair coat.

Finally the anæmia and emaciation become so advanced that the animal dies.

Complication with acute secondary infection can induce high fever and thus bring about death of the animal. Complication with gangrene is often accompanied by persistent fever.

Termination.—Udder-tuberculosis causes a gradual progressive destruction of the udder-tissue, which may require from a month to a year. Destruction progresses rather rapidly during the period of lactation, but more slowly, often very slowly, during the non-lactating period.

Clinical Symptoms.—The condition is a chronic one and usually affects older cows which are 7 years old or older.

One or more quarters are involved and of these at least one of the posterior quarters. The affected quarters are enlarged, especially in the inferior parts. Usually the enlargement is uniform, seldom irregular, in which latter cases nodular foci are present. The enlarged portion is hard and painless. These portions may infrequently present a wound which is chronic and contains a stinking, soft, caseous, purulent secretion.

The lymph-gland of the affected quarter is correspondingly increased in size. Only in one case was the lymph-gland not visibly enlarged.

The quantity of milk is at first slightly but later greatly diminished. The milk is of normal appearance in low-grade alterations of the udder, but gradually changes with the progression of the disease. It becomes yellow and mixed with pus and finally yellowish-gray and purulent. Usually, however, it is thin and contains a few free flocculi of fibrin.

This alteration of the milk can occur rather quickly. After one month the milk can become distinctly changed. Usually, however, this does not occur until after two months. There are cases, however, in which the milk had retained its normal appearance until four months after the inception of the condition. These differences naturally depend upon the degree of distribution and the rapidity of development of the tuberculous lesions.

In the rather infrequent complications with secondary infections or saprophytic invasion the milk can become grayish-red, especially in the more acute secondary infections, and stinking in gangrene.

Upon microscopical examination of the sediment one usually finds a moderate number of acid-fast tubercle bacilli outside of the cells.

With a moderate number of tubercle bacilli I believe that one may find from one to ten bacilli in every field when

a uniform smear of the material is made on the slide and the organism is enlarged 550 times.

The content of tubercle bacilli may vary distinctly, however. There are cases where from 100–200 tubercle bacilli occur in each field (Fig. 24), and also where the tubercle bacilli are exceptionally few, one in every five fields, one in a smear or even less, so that a microscopical diagnosis is made with great difficulty or not at all.

The milk can also contain other acid-fast rods besides tubercle bacilli. They may be acid-fast actinomyces rods in udder-actinomycosis or saprophytic acid-fast rods in pollution of the milk. These rods may be mistaken for tubercle bacilli or, on the contrary, tubercle bacilli may be mistaken for these acid-fast rods, especially in severely contaminated milk samples.

FIG. 24.—Tubercle bacilli in milk sediment. Ziehl (× 550).

Of 841 milk samples examined microscopically here in the High School, tubercle bacilli were demonstrated in 155 cases, or 18 per cent. of the samples.

The following errors were committed: In two cases (0.24 per cent.) no tubercle bacilli were found in obstinate udder-tuberculosis. In one case the milk was examined again at fourteen-day intervals without results. The udder showed a slight distribution of tuberculosis without visible caseation in one anterior quarter. The foci contained a few tubercle bacilli. The milk caused tuberculosis in guinea-pigs. In the second case the milk was only examined once. A possible error in staining is not to be completely disregarded. The udder showed a few tuberculous caverns containing loose caseous necroses which were the size of a nut. In the necrotic areas a moderate number of tubercle bacilli were found. Perhaps more errors of the same kind were made, but they attained a very small number, perhaps fifteen of the samples examined.

In two cases (0.24 per cent.) actinomycotic rods were taken for tubercle bacilli. The udder showed no tuberculosis, but extensive actinomycosis.

In two cases (0.24 per cent.) acid-fast rods were mistaken for tubercle bacilli. The udder was now examined and found to be free from tuberculosis.

In one case (0.12 per cent.) tubercle bacilli were present but mistaken for acid-fast rods, since the sample was very badly contaminated. A new sample was sought, but not obtained, since the cow had been sold in the meantime. A guinea-pig was inoculated with the milk and developed tuberculosis. In at least two other cases the same condition was present, but the tubercle bacilli were found in the new samples obtained.

Later two cases were confused with pyogenes bacilli. The udder, when examined, showed no tuberculosis, but extensive pyobacillosis. Both of these milk samples, according to my report, had presented a few tubercle bacilli, which were very few, however, as only one tubercle bacillus was found in two smears. Both samples contained numerous pyogenes bacilli. Frequently a few pyogenes bacilli were not decolorized and had been mistaken for tubercle bacilli.

The rather quick microscopic diagnosis of udder-tuberculosis gives very good results in spite of a few possible errors.

These errors may be eliminated to a great extent by observing the following rules:

1. The milk samples should only be taken from the affected quarter or quarters. All the milk possible is removed from the diseased quarter; in this way the bacillary content is the greatest. If the quantity of milk is great, the milk sample can stand for half an hour, when the upper part may be poured off and the remainder, which contains

a rich sediment, is sent in for examination. The milk sample naturally should not be strained or filtered.

2. One should avoid precipitation in the milk. Casein precipitation occurs in acid fermentation. This is prevented, especially in summer, when the milk easily becomes sour, by the addition of an antiseptic, such as boric acid, which does not alter the milk. Addition of chloroform is not indicated, since chloroform induces precipitation of the fat. The precipitations cause the smear to be poor in bacilli and lessen the possibility of finding the tubercle bacilli.

3. If the result of the examination is negative a new sample of milk can be obtained after from ca. eight to fourteen days, which often contains more tubercle bacilli which are then more easily discovered.

4. Strict cleanliness should be observed in the collection of milk samples. This reduces contamination with acid-fast organisms.

If the condition is so old and the udder so destroyed that no milk can be obtained, a harpoon probe must be made in order to obtain a diagnosis; moreover, such a sample is unnecessary.

Treatment of these conditions is not permitted in Sweden, according to the Royal Order of May 1, 1903. The animal must be slaughtered if the diagnosis of udder-tuberculosis is established. [No such Federal law exists in this country, but interstate shipment is prohibited. The majority of municipal laws prevent shipment of milk from such a cow into the municipality.]

If the animal is suspected of udder-tuberculosis it can be isolated from the rest of the animals and milked last in anticipation of the diagnosis. Its milk should not be used at all for man and animals, or only after it has been boiled.

Summary

1. Infectious organism: tubercle bacillus.
   Occurrence.
   Resistancy.
   Experimental mastitis.
2. Udder-infection: udder-tuberculosis.
   Stroma-infection.
      Ports of infection: blood, wounds and teat canals.
      Local conditions.
         Primary tubercles.
            Distribution: regional, lymphogenous, hæmatogenous distribution as well as distributed by the milk.
         Growth of tubercles.
            Alteration of necrosis. Colliquation and calcification.
            The tubercles cause stagnation of the blood and the milk.
            Ulceration of the milk-ducts (milk-infection).
            Ulceration of the blood-vessels (blood-infection).
            Ulceration through the skin or into wounds.
         Types:
            1. Primary udder-tuberculosis.
               a. By teat-infection.
               b. By wound-infection.
            2. Secondary or embolic udder-tuberculosis.
         Secondary infections:
               a. By the teat canals.
               b. By wounds.
         Saprophytic invasions:
               a. Milk-gangrene.
               b. Wound-gangrene.
      General conditions:
         Blood-infection.
         Intoxication.
      Termination.
      Intoxication.
      Clinical symptoms.
         Microscopical diagnosis.
         Possible errors.
         Rules for collection of milk samples.
      Treatment.

CHAPTER XI

UDDER-ACTINOMYCOSIS

Infectious Organism

The infectious organism is the actinomyces bovis or ray fungus.

Characteristics.—The pathogenic actinomyces bovis is a small thin filamentous organism which sometimes shows finger-like branching and which forms round spore-like bodies. In the animal body it forms round radiated colonies. Round finger-like or club-shaped bodies are arranged on the surface of the structure in a radiating manner and appear at the edge as a marginal zone. The filaments are difficult to stain, but may be stained by Gram's method. This fungus is exceptionally difficult to cultivate.

Occurrence.—The actinomyces fungus appears to be of uniform geographical distribution.

In Sweden it is quite generally distributed, but appears to be present more especially in certain known districts, however.

It appears to be found on straw and bearded grains, since the infection is often carried by these foreign bodies.

Morphology.—The actinomyces fungus occurs in two forms, filamentous and spherical.

The appearance of the filaments varies. They may form single fine rods which closely resemble the tubercle bacillus, and may also be mistaken for tubercle bacilli. As a rule, however, they are somewhat thicker, show finger-like branching, and the branches often show swollen club-shaped ends.

The spherical form or spore-like bodies (perhaps real spores) are somewhat uniformly round and fairly large, approximately the same size as the staphylococcus.

The fungus forms characteristic colonies in the animal body. These structures are yellow or yellowish-brown, round or irregularly shaped polygonally rounded bodies from 0.1 to 2 mm. in diameter. The larger colonies are always visible to the naked eye as yellow or yellowish-brown points.

With a magnification of 30 diameters they show a homogeneous wax-like appearance and a uniform or usually slightly crenated margin (Fig. 25). With a greater magnification (× 250) the margin presents a zone of radiating club-shaped bodies (Fig. 26).

On cut section and with greater magnification the colonies present a core and a radiated finger-like margin. The centre shows the little round spore-like bodies, and outside, the margin shows the finger-like divisions and club-shaped swollen filaments (Fig. 27). The most external part of the margin formed by the club-shaped rays may be deeply stained with eosin and picric acid. In this way these stains are often of assistance in searching for the colonies.

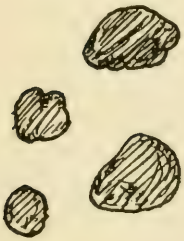

Fig. 25.—Actinomyces colonies in pus. Unstained preparation in saline solution (× 30).

Fig. 26.—Actinomyces colonies in pus. Unstained preparation in saline solution (× 250).

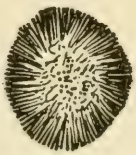

Fig. 27.—Actinomyces colony. Cut section. Gram. (× 300). Sporelike bodies in the centre.

The zone of club formations appears around the filaments like a protective capsule. It only occurs on the periphery and guards against the harmful protective influences of the animal host. The size of these clubs varies

considerably. They are usually small and by magnifying 250 diameters are just barely visible. In less frequent cases they are quite large and may be seen by magnifying 100 diameters.

The fungus is very difficult to stain, is Gram positive, but must be carefully decolorized. The filaments are not infrequently acid-fast.

CULTIVATION.—The fungus is exceptionally difficult to cultivate. I have attempted its cultivation repeatedly without success and therefore advise reference to text-books on bacteriology for its cultural characteristics.

PATHOGENICITY.—Since I did not cultivate the organism I performed no animal experiments.

The actinomyces fungus is a cell parasite like the pyogenes and tubercle bacilli and one may therefore reach the same conclusions in experiments with the actinomyces fungus as with the pyogenes bacillus. One may also expect that the fungus injected into the udder will soon be taken up by the leucocytes and removed from the milk. These leucocytes carry fungus into the udder stroma, where fungus colonies are formed which may destroy the milk-ducts and finally be discharged into the milk after from one to several weeks.

UDDER-INFECTION

This is rare. The actinomyces fungus always produces a typical stroma-infection [interstitial mastitis].

STROMA-INFECTION [*Interstitial Mastitis*]

Like tuberculosis, this infection possesses a typical chronic progressive character.

PORTS OF INFECTION.—The organism can gain entrance through the teat canal, through wounds or through

the blood-stream. I have never seen the infection produced through an udder-wound.

An infection through the blood-stream always follows actinomycosis of the lungs. This disease is rare, however, perhaps less frequent than actinomycosis of the udder. Therefore I maintain that this portal of entry does not play so great a rôle since I know of no positive case of actinomycosis of the lungs and udder together.

The most important port of infection is the teat canal. Of seven actinomycotic udders sent to the Veterinary High School three of the cases were reported to have shown no alterations in the lungs. Lung tuberculosis was observed in one case and in the three other cases no report was made concerning the lungs. None of the udders presented wounds.

Of four cases with complete history, therefore, three were positively primary udder-actinomycosis through teat infection. In the fourth case it may be possible that actinomycosis was also present with the tuberculosis but was overlooked by the veterinary inspector. This case is therefore probably, but not positively, primary udder-actinomycosis through teat-infection.

It is not to be thought that the slowly growing actinomyces fungus develops in the milk in the teat canal, but one must admit, on the contrary, that the organism is mechanically forced into the teat canal on a straw.

Local Conditions.—Leucocytes pick up the fungus and deposit it in the lymph spaces of the stroma, where the fungus finally destroys the leucocytes.

Primary Lesion and Inflammation.—The fungus paralyzes and necrotizes the leucocytes which have engulfed it and this cell necrosis constitutes the primary lesion.

A great number of cells emigrate and collect around the primary lesion (emigratory stage of inflammation), and outside this wall of leucocytes a capsule of new con-

nective tissue forms. This structure constitutes the primary actinomycoma (Fig. 28).

The wall of cells of an actinomycoma often presents two different layers:

1. An internal thin layer of lymphocytes showing little protoplasm and large round nuclei rich in chromatin.

2. An external thicker layer of epithelioidal connective-tissue cells showing oval vesicular nuclei poor in chromatin, but with considerable protoplasm.

In the centre of the focus the fungus gradually develops into a characteristic ray fungus colony. The adjacent part of the zone of cells surrounding the fungus undergoes necrosis, but this is corrected by the emigration of fresh cells to replace them. The formation of actinomyces colonies develops slowly.

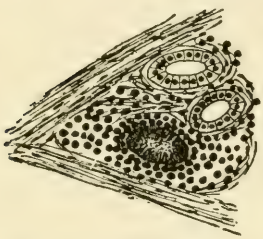

FIG. 28.—Actinomycosis of the udder, stroma-infection (× 150). An oval actinomyces colony with surrounding inflammatory zone of leucocytes is visible in the swollen intralobular connective tissue. The tubules are compressed. The actinomycoma lies in a corner of interlobular connective tissue.

DISTRIBUTION.—The infection shows a tendency toward metastasis. Metastasis in actinomycosis is decidedly less frequent than in tuberculosis.

Extension of the condition is principally regional. By leucocytic transportation a single fungus cell of a colony forms a daughter actinomycoma in the neighborhood of the primary actinomycoma. This local metastasis usually occurs in the direction of the lymph-stream.

Finally the actinomyces fungus can penetrate the lymph-vessels, be carried to the supramammary lymph-glands and thus by lymphogenous metastasis produce new actinomycotic foci. This metastasis to the lymph-glands appears to take a very long time. Of four udders received the lymph-glands were still attached. In only one case was the lymph-gland on the side of the udder affected swollen and actinomycotic. This udder showed very old actinomy-

cotic foci up to the size of a walnut, while the foci in the lymph-glands were the size of a pea.

Metastasis by the milk plays a greater part and will be discussed later. Metastasis by the blood-stream appears to be quite rare. Actinomycosis of the lungs is somewhat infrequent, while actinomycosis of the tongue and jaws is common.

Growth of Actinomycomata.—The primary actinomycoma manifests a very gradual development. The central colony is very slowly enlarged. During the lactation period development is more rapid, but is slower during the quiescent state.

Neighboring actinomycomata, as primary foci, and their daughter foci may coalesce by extension and produce nodules ranging in size from a pea to a walnut or larger. These nodules are composed of soft connective tissue and numerous foci containing evenly distributed actinomycotic colonies.

Secondary Alterations of Actinomycomata.—When great quantities of fluid are present, especially when an actinomycoma has destroyed a milk-duct and thus is brought in direct contact with the milk, the actinomycoma can soften, disintegrate, and become a viscid, purulent, slimy, thick fluid.

Conversely when little fluid is present the actinomyces colonies develop so slowly that the connective tissue of all the actinomycoma proliferates very vigorously, more or less circumscribing the infection by encapsulation. Under such conditions the actinomyces colony can undergo retrogressive metamorphosis, become partially or entirely necrotic and finally be transformed into a concrement by calcification.

Actinomycomata by their development appear to offer an obstruction to the blood circulation and to the milk flow.

Ulceration of Milk-ducts.—Actinomycomata may destroy the wall of a milk-duct. By absorption of milk

actinomycomata may become swollen, softened and disintegrated and finally be discharged into the milk. In this manner the ray fungus can: (1) be discharged into the milk; (2) be spread to other parts of the same quarter by leucocytes; (3) stagnate in the milk-ducts together with softened necrotic parts of the actinomycoma.

Genuine milk-infection [*parenchymatous mastitis*] does not appear to occur; that is the stagnated fungus does not induce necrosis of the subjacent epithelium, at least not to any marked degree. Formation of croupous membranes does not appear to occur in this condition.

Ulceration of a Blood-vessel Wall.—This is possible, of course, but only appears in exceptional cases and with the relative infrequency of actinomycosis of the lungs.

Ulceration Through the Skin.—I have not observed this condition in any of my cases. It is possible, however, like the not uncommon ulceration of the skin in actinomycosis of the jaw. It is certainly very infrequent in the udder, nevertheless.

TYPES

Depending upon the ports of infection the following types are distinguished: (1) primary udder-actinomycosis, (*a*) by infection through the teat canal, (*b*) by infection through wounds; (2) secondary or embolic udder-actinomycosis.

These types have the same general differential characteristics as the previously described types of pyobacillosis (see Pyobacillosis).

Primary udder-actinomycosis following teat-infection is the most common type. The foci are so distributed in the udder that one cannot expect to find an anatomical difference from the embolic type upon post-mortem examination. The condition almost always begins in one quarter, but may frequently be spread to other quarters by

lymphogenous metastasis and by infection through the teat canal. In one case of distinctly primary udder-actinomycosis induced by teat-infection all four quarters became affected.

Primary udder-actinomycosis following wound infection is a condition I have not seen, but if it occurs it certainly has a particular anatomical character which closely simulates the corresponding type of udder tuberculosis.

Secondary or embolic udder-actinomycosis is a condition I have not observed in any of the udders that I have examined, but that it can occur appears to be natural.

The possible secondary infections might be strepto- and staphylomycosis, colibacillosis and perhaps pyobacillosis.

Secondary infection through wounds always occurs through an actinomycotic wound, and here, as with all chronic wounds, secondary infection is the rule. Secondary infections which one may expect in this case are the same as those previously described.

Saprophytic invasion can induce milk- and wound-gangrene.

Milk-gangrene [gangrenous mastitis] does not appear to occur in primary udder-actinomycosis. It is possible in secondary infections.

Wound-gangrene, when it occurs, always develops from an actinomycotic wound, and it may easily develop in this condition. As previously stated, a wound of this kind is very infrequent.

General Conditions.—Infection of the blood with metastasis to the lungs is an uncommon complication.

Intoxication.—Udder-actinomycosis usually presents no distinct general symptoms. If the condition is far advanced, however, or metastatic processes have developed the animal may emaciate nevertheless, and finally present symptoms of anæmia and debility.

Termination.—Udder-actinomycosis is a typical local condition and causes a progressive gradual destruction of the udder, extending over the course of a year.

Clinical Symptoms.—The condition is very chronic. The affection usually begins in one quarter, but it can affect other quarters by extension or by infection through the teat canal. The quarters affected are enlarged, especially in their inferior portions. The increased part is hard and painless.

The lymph-gland of the diseased quarter is swollen if the condition is an old one. Usually it is not enlarged.

The quantity of milk gradually diminishes. The milk is of normal appearance for a long time, even for several months, but gradually becomes thin and mixed with pus and finally yellow and purulent, still continuing somewhat thin.

Upon microscopical examination of the sediment in an unstained coverslip preparation made by careful isolation of the elements of the sediment in physiological salt solution by the use of a needle, usually a very few, seldom many, actinomyces colonies were observed outside the cells. These actinomyces colonies are always very small and very spongy and frequently present a very indistinct radiating form. The yellow color, the rounded crenated margin, the homogeneous waxy condition and the cells are usually of great assistance in finding the colonies with low magnification (ca. 30 diameters).

Diagnosis with the microscope is usually very difficult and errors may easily occur; that is, one may find no actinomyces colonies after diligent search in spite of the fact that udder-actinomycosis is present. I have had one case of this kind.

Preparations stained after the method of Gram also gave uncertain results. In preparations stained by the acid-fast method one can sometimes find a few fine acid-

fast rods which greatly simulate tubercle bacilli and which may also become confused with them.

The PROGNOSIS is unfavorable for the udder. The condition continues to a progressive destruction of the udder.

TREATMENT.—Prophylaxis is the most important. All open cases of actinomycosis of the jaw, tongue or udder spread the infectious organisms and in this way encourage the possibility of actinomyces infection and thus udder-actinomycosis. Slaughter of all animals showing open actinomycotic lesions is the safest measure to adopt in prevention of such an infection. If the owner will not consent to this the animals are isolated as strictly as possible and so treated as to disinfect the diseased products bearing the infectious organisms.

Actual Treatment.—One should endeavor to induce sclerosis and atrophy of the infected part of the udder wherever possible, which prevents progressive destruction and encourages healing.

The cow remains and the entire infected quarter is deeply point-fired with a cautery iron, which promotes sclerosis and contraction to a high degree.

The treatment is completed by administering 8 to 12 g. of potassium iodide per os daily for from three weeks to a month, a course which is always indicated in actinomycosis.

SUMMARY

1. Infectious organism: actinomyces bovis.
   - Characteristics.
   - Occurrence.
   - Morphology.
     - Filamentous form.
     - Club form.
     - Colonies.
   - Staining.
   - (Cultivation and pathogenicity.)

2. Udder-infection: Udder-actinomycosis.
   Stroma-infection [interstitial mastitis].
      Ports of infection: teat canals, wounds and blood.
      Local conditions.
         Primary actinomycoma.
         Metastasis: regional, lymphogeneous and hæmatogenous metastasis as well as galactogenous metastasis.
         Development of actinomycomata.
         Secondary alterations of actinomycomata.
            Softening and encapsulation.
         Ulceration of milk-ducts.
         Ulceration of blood-vessels.
         Ulceration through the skin.
         Types:
            1. Primary udder-actinomycosis.
               a. By teat-infection.
               b. By wound-infection.
            2. Secondary or embolic udder-actinomycosis.
         Secondary infection.
            1. By the teat canal.
            2. By wounds.
         Saprophytic invasion.
            1. Milk-gangrene.
            2. Wound-gangrene.
         General conditions.
            Blood-infection.
            Intoxication.
         Termination.
         Clinical symptoms.
         Prognosis.
         Treatment.
            Prophylaxis.
            Actual treatment.

CHAPTER XII

UDDER-NECROBACILLOSIS

I HAVE very little to say concerning this rare affection of the udder since I have never seen a case. Jensen reports a case (Mastitis in Animals, 1899). Lucet's Case VIII (Mammites Aigues, 1891) is perhaps this infection, although negative results were obtained by inoculation of dogs, which was probably due to too short observation. On the other hand, the bacteria isolated by Steiger as necrosis bacilli (Bacterial Findings in Mastitis, 1904) were certainly not bacillus necrophorus, since they were Gram positive, but were probably pyogenes bacilli.

The infectious organism is the necrophorus bacillus. It is a somewhat fine, Gram negative, obligate anaërobic, thread-like bacillus which is very difficult to cultivate. It is pathogenic for mice and dogs and produces typical chronic lesions following inoculation.

The organism appears to be a typical cell parasite, like the pyogenes and tubercle bacillus.

UDDER-INFECTION

The bacillus necrophorus produces a distinct typical stroma-infection [interstitial mastitis] of chronic progressive character as in the three previous udder-infections, and the milk-infection [parenchymatous mastitis] is only a complication of it.

PORTS OF INFECTION.—The infection can take place through the blood-stream (Jensen) and probably through the teat canal and wounds.

TYPES OF THE LOCAL CONDITIONS.—The case reported by Jensen was an embolic udder-necrobacillosis.

Lucet's case, which was only described clinically, was perhaps a primary udder-necrobacillosis through teat infection. No case of primary udder-necrobacillosis through wound infection has been reported in the literature available.

Mixed Infection.—In the case described by Lucet streptococci were found in the exudate in addition to these bacilli.

CHAPTER XIII

CLINICAL DIAGNOSIS OF MASTITIS

Mastitis Caused By External Force

Bruises and wounds of the udder are easy to diagnose.

The sclerosis following repair of an abrasion might possibly be mistaken for a chronic udder-infection. A good history and careful examination of the milk are of great assistance in making a differential diagnosis.

Udder-infection

Infections of the udder may be divided clinically into wound-infections and specific udder-infections.

INFECTED UDDER-WOUNDS

These wound-infections are recognized by a more or less severe swelling in the neighborhood of the wound, and the secretion, which usually consists of yellow pus, but which may infrequently present a red-colored serous fluid in severe acute infections.

Wound-infections may be divided into acute and chronic forms, depending upon the course.

The acute wound-infections are streptomycosis, staphylomycosis, and colibacillosis.

Wounds complicated by acute infections usually heal in ca. one month. The infection often begins with fever. At first the secretion is serous, but later becomes purulent. If the infection advances when the wound is fresh it may become complicated with an acute milk-infection. Gangrene of the wound secretion is an exception.

A clinical differentiation of the three types appears to

be impossible and is of no practical significance, since wound-infection is seldom treated as a single infection, but as a mixed infection.

The chronic wound-infections are pyobacillosis, tuberculosis and actinomycosis.

These complicated infectious wounds show little or no tendency to heal, but gradually become larger.

Fever can occur in complications with acute infections (mixed infection). In gangrene, fever is always present, otherwise these infections are free from fever.

All these chronic infections spread into the udder-tissue, which often manifests a widely circumscribed hard swelling around the wound, together with alteration of the milk (see under Specific Udder-infections).

In *pyobacillosis* the wound-secretion is thick and yellow, the consistency of cream or salve, and contains no calcareous granules. The walls of the wound are composed of smooth, firm and tough connective tissue, which is colored green in gangrene by pigment of putrefaction. Gangrene is very common. The corresponding lymph-gland is not enlarged.

In *tuberculosis* the secretion is yellow, soft, caseous and frequently contains calcareous granules which are easily felt by rubbing the pus between thumb and finger. The walls of the wound are often irregular and nodular with tubercles. Gangrene is very common. The corresponding lymph-gland is enlarged.

In *actinomycosis* the quantity of the secretion is usually small, qualitatively it is yellow and viscid, due to disintegration of actinomycomata. The wound may become filled with red, soft, spongy granulation tissue which may protrude from the wound as a great oval red nodular actinomycoma. The wound does not heal, but gradually increases. Gangrene is not so common because of the small quantity of the secretion. The corresponding lymph-gland is usually not enlarged.

GENUINE UDDER-INFECTIONS

These infections are manifested by changes in the udder and the milk. They may also be divided into acute and chronic forms.

The *acute genuine udder-infections* are streptomycosis, staphylomycosis, and colibacillosis. They are severe in the beginning, but are seldom fatal. Usually repair is established by inflammation and sclerosis in a month.

They are typical infections [parenchymatous mastitis] and usually begin in one quarter, but may also spread to other quarters by infection. Simultaneously with the infection alterations in the milk occur. In severe infections the milk is transformed into a serous, even red-stained, thin, somewhat muddy fluid secretion. On the other hand it may consist of thick yellow pus or a thick yellow mixture of pus and milk. Slimy flakes of fibrin are frequently present in the pus or altered milk.

In addition to the changes in the milk a hard swelling of the quarter occurs at the same time, which is at first painful and later becomes painless and may vary to a high degree. The corresponding lymph-gland is but slightly or not at all affected. The condition usually begins with fever, which subsides after a few days. Complications with acute secondary infections and gangrene produce a recurrence of fever and destroy the healing process. These complications are rare, however.

A specific clinical diagnosis of these affections is usually impossible.

One may suspect streptomycosis if the condition is epizoötic and affects a number of cows with a high fever of short duration, and high or low grade disease processes in the udders and milk. This is especially so when the joints (tarsal joint) synchronously present a painful swelling. If the condition is vigorous and severe or fatal, one

may suspect staphylomycosis. The more common sporadic cases may be any of the three infections.

The chronic genuine udder-infections may be divided into two groups:

1. Atypical chronic udder-infections which begin as severe acute infections and then become chronic.

2. Typical chronic udder-infections which are not noticeable in the beginning.

Atypical udder-infections are undoubtedly streptomycosis.

The atypical chronic streptomycosis begins with all the symptoms of a severe acute udder-infection (milk-infection) [parenchymatous mastitis] with alterations of the milk, swelling of the udder and fever. The fever subsides, but the alterations of the milk and swelling of the udder remain about a month. The udder finally becomes as hard as stone, through sclerosis, and may manifest nodular foci which, when incised, present pus and probably firm necrotic sequesters in an abscess cavity surrounded by a smooth connective-tissue wall.

Pyobacillosis and tuberculosis may run a course like an atypical chronic udder-infection when they enter an acute udder-infection as complicating secondary infections. In these cases the acute infection is simply displaced by a different chronic infection.

Typical chronic udder-infections are not noticeable in the beginning, but gradually produce alterations in the milk and udder.

When a blood-vessel is injured the milk may be slightly tinged with blood. The lesions of the udder are but slightly or not at all painful.

These infections may be divided into two sub-groups: (*a*) those which cause a distinct swelling of the udder; (*b*) those in which no distinct swelling is present.

The typical chronic udder-infections which cause a dis-

tinct swelling of the udder are the typical stroma-infections: Pyobacillosis, tuberculosis, and actinomycosis.

The swelling of the udder is the first distinct symptom of the disease. At first the milk is not changed, then it becomes mixed with pus and finally, in about a month, purulent.

In order to observe the lesser changes in the milk, the diseased quarter is well milked out and the milk poured into a litre flask made of uncolored glass. The flask is stoppered and then inverted for ten or fifteen minutes. At the end of this time the sediment has collected in the neck of the flask and on the stopper, so that one may readily examine it for quantity, color and other qualities.

Pyobacillosis usually affects but one quarter, but may spread to other quarters by infection. It produces a moderate and finally large hard swelling of the udder. The corresponding lymph-gland is not swollen.

The milk frequently has a normal appearance, but contains pus. It gradually becomes more intensely altered and finally, after months, the milk becomes purulent, thick and pulpy in consistency, due to the presence of thicker pus. Gangrene is a common complication and imparts a foul odor to the milk, which assumes a characteristic stink, specific of decomposed cabbage.

If the condition is an old one abscesses may form in the udder which contain yellowish-green, thick, pulpy pus. These may rupture upon the external surface and produce fistulæ.

The condition is free from fever in the beginning, but it occurs following the frequent complications with acute secondary infections and gangrene.

Tuberculosis attacks one or more quarters. The swelling is very large and hard. The corresponding lymph-gland is always enlarged and sometimes very greatly so.

The size of the lymph-glands is different in the different

cows, but in the same animal both supramammary lymph-glands are the same size under physiological conditions. Thus a unilateral lymph-gland swelling is easy to determine, while, on the other hand, bilateral swelling is often difficult.

At first the milk is normal in appearance, later becomes mixed with pus and finally, after a period of several months, is purulent. In this condition it is composed of a yellow, serous, thin pus containing flakes of fibrin. Milk-gangrene is exceptionally rare.

Secondary infections and abscess formation are very infrequent. The contents of the abscesses when they do occur is made up of yellowish-white, soft, caseous pus containing calcareous granules. The condition is not associated with fever.

Actinomycosis attacks one quarter, but may extend to other quarters. The enlargement is not great and develops slowly. The corresponding lymph-gland is usually not enlarged (only in very old udder-actinomycosis is there any great enlargement).

The milk is normal in appearance for more than a month, but finally becomes yellow and purulent. It is then a yellow, serous, thin fluid pus containing flakes of pus. Milk-gangrene is exceptionally rare. Secondary infections and abscess formation are rare and the condition is not accompanied by fever.

Typical udder-infections which present *no distinct swelling of the udder* are caused by streptococci.

Typical chronic streptomycosis is a milk-infection [parenchymatous mastitis]. It usually affects one quarter, but may spread to the other quarters by infection.

The milk is changed gradually. It becomes yellow, thick as cream, and finally almost pulp-like in consistency, and contains a great number of floating pus-cells. Milk-gangrene is exceptionally rare.

Ultimately the udder becomes altered. It does not collapse after milking, especially the parts around the base of the teats, and is firm or hard, but not distinctly enlarged. These changes slowly extend laterally and upward. The supramammary lymph-glands are not affected. Secondary infections are rare. Abscesses do not occur. The condition is free from fever.

Microscopic Examination of Udder-infections.—This corroborates or corrects the clinical diagnosis.

The material to be examined is the wound secretion in wound-infections and the sediment of the milk in genuine udder-infections. In these examinations one may expect to find great numbers of pyogenes bacilli in the pathologic products. Conversely the tubercle bacilli and actinomyces colonies are only present in small numbers.

In acute milk-infections [parenchymatous mastitis] great numbers of bacteria are present during the first few days, but later become few. In typical chronic streptomycosis the bacteria are very numerous.

In cases where the cocci appear in the diplococcus form in coccus infections a diagnosis of staphylomycosis or streptomycosis may be very difficult.

A uniformly round shape and many single cocci point toward staphylococci, while an oval shape and the absence of single cocci emphasize streptococci. If in the first case the cocci are grouped in grape-like bunches and in the last case in chains, and capsules are present, the diagnosis is certain.

Summary

1. Mastitis caused by external force.
   Udder-bruises and udder-wounds.
2. Udder-infections.
   A. Macroscopic diagnosis.

1. Infected udder-wounds.
   a. Acute wound-infection.
      Streptomycosis.
      Staphylomycosis.
      Colibacillosis.
   b. Chronic wound-infection.
      Pyobacillosis.
      Tuberculosis.
      Actinomycosis.
2. Genuine udder-infections.
   a. Acute genuine udder-infections.
      Acute streptomycosis, staphylomycosis, and colibacillosis.
   b. Chronic genuine udder-infections:
      aa. Atypical chronic udder-infections:
         Atypical chronic streptomycosis (which, together with pyobacillosis and tuberculosis, complicates acute infections).
      bb. Typical chronic udder-infections.
         1. With distinct udder swelling.
            Pyobacillosis.
            Tuberculosis.
            Actinomycosis.
         2. Without distinct udder swelling.
            Typical chronic streptomycosis.

B. Microscopic diagnosis.
   Material for examination.
   Quantity of bacteria in pyobacillosis, tuberculosis and actinomycosis, in acute milk-infections and in typical chronic streptomycosis.
   Differential diagnosis between streptococci and staphylococci.

Streptomycosis is the most variable of all the genuine udder-infections since it can have at least three different clinical forms.

In general a careful examination of the animal, together with a good anamnesis, can enable one to arrive at a comparatively correct diagnosis.

The length of the course corrects or corroborates our diagnosis.

CHAPTER XIV

AUTOPSY

Post-mortem Technic

The udder, together with the supramammary lymph-glands, is dissected from the abdominal wall and divided into its two lateral halves with a knife.

The halves are placed in a certain definite position on a table or other suitable surface in order to facilitate observation of the teats and lesions and remembrance of their topography. Each half is placed upon its median surface, bringing the lateral or external side uppermost, teats proximal of the autopsist and abdominal margin distal. Thus the fore quarter of the left half is at the left and the fore quarter of the right half is naturally at the right (Fig. 29).

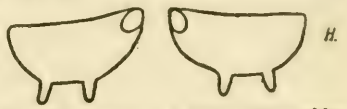

Fig. 29.—Position of halves of the udder at autopsy. *L.* = left. *H.* = right

External Examination.—The normal udder-half lies limp, collapsed and flattened out upon the table. The half is somewhat thin and its inferior margin fairly sharp. The consistency of the udder is soft and elastic.

One looks for wounds and other lesions and observes their position, size, number, consistency, degree and distribution. If milk is present a little should be milked into the hand from each quarter. If the milk from any quarter is altered a sample should be collected from each diseased quarter.

Lymph-glands.—These are examined *in situ*. Their size under physiological conditions is not constant, though the diameter is usually 6 to 9 cm. Their size is commonly indicated by the terms very greatly, moderately, slightly or not at all enlarged.

The lymph-glands are then divided by a longitudinal incision, the halves laid apart and the structure examined for lesions. One should carefully observe whether the incision be dry. Normally it is moderately dry. When juicy it indicates œdema. It may be hard from induration. If any of these conditions be present transverse incisions are made through them.

If a teat is affected it should be amputated close to its base and the cistern and teat canal examined after its ablation.

Cut Surface of the Udder.—Each udder-half is now laid open like a book by making a sagittal longitudinal incision parallel to the table and extending to the cisterns.

Under physiological conditions the incision is more or less juicy and the juice in the incision consists of milk. The cut surface bulges outward so that it is slightly convex. It presents a fine net-like framework of white connective tissue which divides the structure into an inestimable number of closely associated flaccid rounded parts ranging in size from a lentil to a pea, called lobuli. Within the lobuli one can see the milk-filled tubuli as exceptionally small, just barely visible white points called milk points. During the lactation period the lobuli are grayish-white and during the quiescent period they are often yellow. The consistency is soft, elastic, tough, and it resists tearing or crushing. More or less milk is found in the cisterns and milk canals, and their mucous membranes are white and uniform.

Under pathological conditions the cut surface may present many different alterations. The juice in the incision may consist of pus or other pathological products. If the cut surface is even, not convex, it points toward diminished elasticity following sclerosis or other changes. If the consistency of the cut surface is firm but pliable, it indicates engorgement and distension of the elastic gland vesicles with firm contents, such as fibrin or cells. In the same

manner the consistency may be soft and elastic, like India rubber, when the vesicles are filled with water. If they are filled with firmer substances as of pulpy consistency the cut surface will be firm but friable. Upon application of pressure to the diseased lobuli pus may be expressed from the incised collecting ducts. If the milk points have disappeared from a number of lobuli in a lactiferous udder it indicates a diminution or suppression of milk secretion, thus diminution of the volume or destruction of the tubuli. One may find abnormal contents such as blood or pus in the cisterns and milk-ducts.

If the condition is extensive, radiating, transverse incisions are made through the lesions down to the inferior side.

Post-mortem Diagnosis

Upon external examination one may possibly observe the presence of wounds or other perceptible lesions, such as induration.

UDDER-WOUNDS

Infected wounds are important on post-mortem examination.

The Post-mortem Diagnosis.—Infected wounds are easily determined. The rule is that all open and moistened wounds are infected. If it contains a yellow or grayish-red or more or less thick pathologic fluid it may positively be designated an infection. If the secretion stinks, gangrene has complicated the infection.

A deep incision is made through the wound, so that the surfaces may be separated and properly examined with the eye. (This incision is usually a transverse incision through the udder.)

If the walls of the wound are red an acute infection is indicated. This may be a simple streptomycosis, staphylo-

mycosis or colibacillosis or a mixed infection which is more common. This can be determined with the microscope.

The wound secretion can be serous and red or grayish-red or thick fluid yellow pus. The walls of the wound are firm and more or less thick. The inner side is smooth, uneven or warty.

If the walls of the wound are not red, but white, or green from gangrene, a chronic infection is indicated. It may be pyobacillosis, tuberculosis or actinomycosis.

These wounds present the same appearance as described in the previous chapter on clinical diagnosis. In addition one sees the specific lesions of these diseases in the cut surface, walls of the wound and in the adjacent tissue.

SOLIDIFICATION OF THE UDDER

These lesions can be produced by bruises or by infection.

An intense red coloring of the cut surface is indicative of a fresh bruise or an acute infection.

Fresh Udder Bruises.—The udder shows a hard circumscribed swelling. The incision is juicy, and the juice of the incision consists of unaltered milk or milk partly mixed with blood. The cut surface likewise presents a circumscribed altered area which is even, firm but friable. It is red in color, but shows a distinct outline. A few lobuli are red, due to blood infiltration following hemorrhage, others are yellowish-gray and show no milk points. The cisterns and milk-ducts contain unaltered milk or milk mixed with blood.

Acute Udder-infections.—These concern the milk-infections: parenchymatous mastitis, acute streptomycosis, staphylomycosis and colibacillosis.

The quarter is more or less swollen and firm. The incision is juicy, and the juice consists of yellow pus. The entire cut surface or only its inferior portion is even but

friable. The color is grayish-red, due to infiltration with blood. Here and there one may see circumscribed dark red areas due to hemorrhage, or circumscribed yellow, firm or pulpy areas of necrosis. The lesion is distinct and no decomposition of the blood is present. The milk points are absent. Yellow pus or yellow fibrin cylinders are expressed upon application of pressure to the lobuli. Yellow pus or yellow firm masses of fibrin are found in the cisterns and in the milk-ducts.

Complication with gangrene is easily determined by the odor.

A macroscopic differential diagnosis of these conditions is usually impossible, although firm, croupous membranes point toward streptomycosis and a decided purulent softening of croupous membranes toward staphylomycosis.

Staphylomycosis can be very severe and fatal, however, in which case the udder shows diffuse necrosis (severe primary lesion) without inflammation. This post-mortem picture is different and very characteristic.

The affected quarter is distinctly swollen and hard. The incision is juicy and the cut surface presents a red serous fluid. The cut surface is even and its consistency is firm but friable. Here and there it shows black and red circumscribed areas due to blood infiltration and hemorrhage. The cut surface is red and rich in blood for the most part. This lesion is very distinct in consequence of a uniform distribution of the red coloring matter, which is not confined to the injected blood-vessel or the areas infiltrated with blood, but also uniformly stains the connective tissues, parenchyma, mucous membranes and secretions. [Imbibition of liberated hæmoglobin following decomposition of red blood cells.] The milk points in the lobuli are entirely absent. Pressure upon the lobuli causes a red fluid to be expressed. Twig-like cylindrical red-stained masses of fibrin can be

removed from the cisterns and milk-ducts. Gangrene, which is a common complication, produces a stinking odor in the udder.

The acute infections, as secondary infections, may invade the chronic udder-infections, yet these are usually easy to recognize by their particular post-mortem characteristics (see later).

A cut surface, but slightly or not at all red-stained, indicates (1) a chronic infection or (2) the healing process of a bruise or of an acute infection.

The chronic infections manifest their individual characteristic post-mortem picture. From the autopsy point of view they may be divided into typical stroma-infections and typical milk-infections.

The typical stroma-infections are pyobacillosis, tuberculosis and actinomycosis. They are usually easy to recognize. If the condition is very early the diagnosis may be difficult and the microscope must be used.

Pyobacillosis.—The affected quarter is hard and moderately swollen. The lymph-glands are not changed.

Upon incising the lesion, the knife is smeared by a yellowish-green, thick, caseous mass of salve-like consistency. The cut surface is even and yellowish-gray. Its consistency is firm. Here and there one observes variable-sized cavities containing yellowish-green, caseous masses, which are surrounded by thick connective-tissue walls, the internal side of which are smooth. The yellowish-gray lobuli show no milk points, but not infrequently a few or more small yellow, sharply defined necrotic or caseous points. Pressure upon the lobuli causes yellow, caseous cylinders to be expressed. Numerous milk canals are distended with these yellowish-green caseous masses. Their walls have become thick, white connective-tissue structures transformed into fistulæ. In other milk canals and in the cisterns one may observe spongy croupous membranes. The

mucous membranes in the cisterns may show small, red, pointed growths (condyloma). In the common complication of gangrene a stinking odor is present.

Tuberculosis.—The affected quarter is usually hard and severely swollen.

The corresponding lymph-gland is increased in size. The incision in the lymph-gland is either succulent, as in udder-tuberculosis, or more or less dry and often gritty, as in advanced udder-tuberculosis. The juicy cut surface is spongy in consistency and shows few, if any, small caseous foci. The dry cut surface is caseous for the most part and therefore yellow in color. The consistency is firm and in calcification very firm.

The incision in the udder is comparatively dry and in advanced tuberculosis gritty, due to calcification. The affected inferior portion of the cut surface is even, the consistency is firm and the color grayish-yellow. The superior portion of the cut surface bulges or is convex. It shows a number of yellowish-gray, round, protruding nodules, varying in size from a poppy seed to a hemp seed, or small wart-like structures situated here and there in the lobuli. The lobuli present no milk points, but small yellowish-white caseous points instead. Usually, but not invariably, yellow caseous masses may be expressed by pressure upon the lobuli. In addition, if the condition is old, one may see yellow, round, sharply circumscribed necrotic foci of variable size ranging from a hemp seed to a walnut or larger. The necrotic parts are loose and thickly fluid or firmly caseous. It can be calcareous and the calcification begins in the centre and is manifested by a brighter point or nucleus which later enlarges. The necrotic area is usually surrounded by very thin connective tissue. In the superior convex part of the cut surface the color is whitish-gray. The consistency is very soft and elastic. The lobuli are

increased in size and the milk points very prominent, due to milk stasis.

In the cisterns and milk ducts one can observe faintly yellowish colored milk, or in advanced tuberculosis yellowish croupous membranes. Here the mucous membrane may also present small, round, crateriform tuberculous wounds. In simple or uncomplicated tuberculosis, gangrene never occurs.

ACTINOMYCOSIS.—The affected quarter is hard and usually enlarged to a lesser degree. The corresponding lymph-gland is usually not involved. Only in very old udder-actinomycosis is it enlarged, when on section it presents grayish-yellow, protruding, round, soft, actinomycotic foci.

The incision in the udder is somewhat dry. The cut surface is even in the affected inferior portion. Here and there one may see sharply defined, round, distinctly protruding or convex yellowish-gray, soft, elastic foci ranging in size from a poppy seed to a hemp seed and larger. Within these foci one finds a number of smaller, yellowish-white, white to brown granules or actinomyces colonies, which are hard and sharp to the touch, due to calcification, if the condition is old. The surrounding parts are grayish-white, without milk points and of tendon-like consistency due to sclerosis.

In the milk canals and cisterns one finds wounds from the walls of which grow yellowish-gray, round, tumor-like spuds of granulation tissue from the size of a pea to that of a bean, which contain yellowish-brown actinomyces granules. Simple actinomycosis shows no tendency toward gangrene.

The chronic milk-infections are only streptomycosis. From the post-mortem point of view as well as the clinical standpoint these chronic streptomycoses may be divided into

atypical chronic and typical chronic streptomycosis. These forms, however, are not sharply separated, but present transition forms.

Atypical Chronic Streptomycosis.—The affected quarters are usually moderately swollen and hard. The lymph-gland is not involved. The incision is dry. The cut surface is even and uniform. The color is grayish-white and the consistency is tendinous and sclerotic.

The lobuli present no milk points. They are whitish-gray due to sclerosis or yellowish-gray from inflammation. Fibrin plugs are expressed by pressure upon the yellowish-gray lobuli. There are yellow, firm and tough croupous membranes in the milk-ducts and cisterns. At the same time one may observe grayish-white, somewhat firm necrotic sequesters in the cut surface which are the size of a pigeon's egg or larger, show an irregular external surface and contain signs of udder-tissue. As a rule the sequester is moistened with a small quantity of yellowish pus and surrounded by a thick white connective-tissue wall.

Typical Chronic Streptomycosis.—The affected quarter is not enlarged, but frequently diminished in size, yet the quarter is not collapsed and thin, and it is somewhat thicker, especially at the base of the teats, but the volume is diminished, however. The consistency is somewhat firm.

The incision is juicy and the juice consists of yellow, thick, cream-like milk mixed with pus. The cut surface is even, whitish-gray and tendinously firm, at least in the inferior parts around the base of the teats. A little above, the cut surface is yellowish-gray, firm and somewhat friable, due to inflammation. The altered lobuli present no milk points, but thick, yellow, cream-like milk can be expressed from them by applying pressure. The superior part of the cut surface presents unaltered lobuli which show milk points. The milk-ducts and cisterns contain yellow, thick, cream-like milk.

Udder-bruises and acute infections in the state of healing may be mistaken for chronic streptomycosis. The following characteristics are important for a differential diagnosis:

Udder-bruises in State of Healing.—The udder presents a nicely circumscribed hard swelling. The cut surface in this part is even, whitish-gray and of tendinous consistency. In the other portions both the udder and the milk are unchanged.

The Acute Infections in the State of Healing.—The udder is somewhat swollen and hard. The incision is succulent, and the juice consists of a mixture of unaltered milk and yellow pus. The cut surface is usually convex and the consistency somewhat soft and elastic. Here and there among the normal lobuli there are groups of yellowish-gray non-protruding lobuli without milk points, which are of firm but friable consistency. With pressure applied to these altered lobuli pus is expressed.

Summary

1. Post-mortem technic.
   - Ablation of the udder.
   - Position for examination.
   - External examination.
   - Lymph-gland.
   - Teats.
   - Cut surface of the udder.
2. Post-mortem diagnosis.
   - A. Infected wounds.
     1. The walls of the wound when red colored, streptomycosis, staphylomycosis, colibacillosis.
     2. The walls of the wound when not red colored, pyobacillosis, tuberculosis, actinomycosis.
   - B. Solidification of the udder.

1. Severe redness of cut surface.
   a. Fresh udder-bruises.
   b. Acute udder-infections.
      aa. Inflammation.
         Acute streptomycosis, staphylomycosis, colibacillosis.
      bb. Primary lesion, diffuse udder-necrosis.
         Staphylomycosis.
            Possibility of the acute infections being complications of the chronic infections.
2. Cut surfaces which are faintly or not at all red.
   a. Chronic infections.
      aa. Typical stroma-infections.
         Pyobacillosis.
         Tuberculosis.
         Actinomycosis.
      bb. Typical milk-infections.
         Atypical chronic streptomycosis.
         Typical chronic streptomycosis.
   b. Bruises and acute infections in healing stages.
      aa. Bruises in state of healing.
      bb. Acute infections in healing.

CHAPTER XV

IMPORTANCE OF MASTITIS TO MILK CONTROL

If the milk is to be used as food for man it must have a normal appearance and must contain no bacteria which are pathogenic for the human family. If the milk is mixed with blood, due to *bruising* of the udder, it is unfit for use as a human food. Naturally, however, the milk from the healthy quarters may be utilized.

The udder-infections are more important. Streptococci, staphylococci, colon bacilli, tubercle bacilli and actinomyces fungus cause disease in man. Of the infectious organisms of the udder of the cow the pyogenes bacillus alone is not concerned in the production of disease in man so far as is known. Caution demands, however, that it be considered dangerous to the human family until the contrary has been proved.

The Following Rules are Indicated.—All milk from the infected quarter is unfit for use as food for man.

Even if the other quarters of the udder are free from the infection, the milk from the infected quarter can easily get into the milk from the healthy quarters during milking in spite of every precaution. Therefore the milk from the healthy quarters should be pasteurized or boiled before it is used, as a precautionary measure.

To prevent contamination of the milk of the other cows the diseased animal must be isolated and milked last. For the same reason the milk from the diseased quarter must be disinfected so as to avoid unnecessary increase in the pathogenic bacterial flora of the cow stable.

When the infection disappears and the milk becomes normal again it may be used without boiling or pasteurizing and the rules concerning isolation may be discarded.

In acute udder-infections where the changes in the milk occur simultaneously with those in the udder, these rules are easy to follow. These infections may be recognized by the attendants without the assistance of a veterinarian.

The udder is considered healthy when it is no longer swollen and hard, and when the milk of a sample milking presents a completely normal appearance and contains no fibrin or pus flakes. This test is carried out by the veterinarian.

In typical chronic infections the above rules are difficult to observe, since the milk in these infections retains its normal appearance weeks or months after the infection has taken place. The acute infections can be recognized by the attendants while, on the contrary, the typical chronic cases can only be diagnosed by the veterinarian.

If a milk-test record is kept, one can take note of all cows which show a premature and rapid diminution in milk secretion and then examine their udders and in this way, perhaps, make a timely diagnosis. If no milk record is kept one is compelled to examine all the udders.

The examination is best made after milking, when the udders are relaxed and collapsed. In this condition a swelling or thickening is more easily determined. The udder is inspected from behind and from both sides. If lesions are observed in this way a closer examination of the udder is made. The lesion is palpated, as are the lymph-glands, and if a chronic condition is suspected in the udder examination of a milk sample is ordered.

The milk sample should only be taken from the diseased quarter. A microscopic examination of the sediment of the milk sample confirms or contradicts the tentative diagnosis.

If a chronic udder-infection is diagnosed one is governed by the rules designated, except in udder-tuberculosis, in which condition the animal must be slaughtered as unfit

for use as a milch cow, according to our [Sweden] statutory regulations. [No Federal law in this country compels an owner to kill such an animal. Law prevents interstate shipment. Most municipal laws prevent sale of milk from such a cow in the municipality.]

If the examination establishes no definite diagnosis or does not dispel the suspicion of a chronic infection, one may proceed as before stated on the history of the animal until an examination, eight to fourteen days later, confirms the diagnosis or completely removes all suspicion of the case.

Such an examination must be undertaken at least once a month in order to constitute an effective milk control. To establish a certainty that none of the milch cows have udder-tuberculosis these examinations must be repeated every fourteen days.

If the milk is to be used as food for calves or pigs one should follow the same general principles.

These animals may be fed the milk from cases of chronic mastitis, if it is of normal appearance and providing it is boiled before being used.

Summary

1. Milk as food for man.
   A. Bloody milk.
   B. Milk containing infectious organisms.
      1. Rules:
         a. Milk from an udder-infection is unfit for use by man.
         b. Milk from the other quarters can be used when boiled.
         c. The diseased cow is isolated and milked last.
         d. The infected milk is disinfected.
         e. The rules are cancelled when the infection disappears.

2. Application of rules:
   a. Acute infections.
      Diagnosis.
      Test to determine disappearance of infection.
   b. Typical chronic infections.
      Diagnosis.
      Slaughter of animals.
      Suspected chronic infections.
      Time between examinations.
2. Milk as food for animals.

The chronic udder-infections have a great economic significance in regulating milk control. The animals may produce a considerable quantity of milk which can be of normal appearance, while at the same time this milk cannot be used as food for man.

These infections are very common as may be seen by the following figures compiled at an abattoir: In the year 1905 there were inspected in the abattoir at Malmo 6936 cows, of which 1042, or 15 per cent. were shown to be affected with mastitis (annual report of the Malmo Abattoir for 1905, edited by A. M. Bergman, director).

These cases of mastitis or udder-infection in abattoirs are nearly always chronic. The acute infections are seldom seen during the quiescent state of the udder, and cows are only slaughtered in the lactation period when circumstances demand immediate slaughter, which is very rare with acute udder-infections in abattoirs.

These cases of mastitis, or better, the chronic udder-infections in the abattoirs, are chronic streptomycosis or pyobacillosis. Udder-tuberculosis or udder-actinomycosis is usually listed in the abattoir records under the head of tuberculosis and actinomycosis, not as mastitis. In the report referred to above the cases of udder-tuberculosis were computed with the figures on mastitis.

Fifteen per cent. of the cows had been affected with chronic udder-infections.

The same report in 1905 showed 155 cases of udder-tuberculosis, which is ca. 2.2 per cent. of the number of cows slaughtered.

Figures from the journal of the Veterinary High School for

examination of udder-tuberculosis show us the occurrence of chronic udder-streptomycosis, udder-pyobacillosis and udder-actinomycosis.

In 1905 and 1906 there were observed:

Streptococci in	192	milk samples
Pyogenes bacilli in	56	milk samples
Actinomyces fungus in	10	milk samples
Total	258	milk samples

Taking these figures as a standard the above-mentioned cows had:

ca. 9.5 per cent. Chronic udder-streptomycosis.
ca. 2.8 per cent. Udder-pyobacillosis.
ca. 0.5 per cent. Udder-actinomycosis.

Total, 12.8 per cent.

If the percentage of udder-tuberculosis be added we have the following figures: The 6936 cows slaughtered in Malmo in 1905 showed:

ca. 9.5 per cent. Chronic udder-streptomycosis.
ca. 2.8 per cent. Udder-pyobacillosis.
ca. 2.2 per cent. Udder-tuberculosis.
ca. 0.5 per cent. Udder-actinomycosis.

Total, 15 per cent. Chronic udder-infections.

These figures suggest how common such udder-infections are. One should assume that these figures apply to every standard of cow, but should consider that the statistics were compiled in an abattoir and that they only include animals slaughtered for various reasons such as old age and disease.

According to the estimate of a practical farmer ca. 16 per cent. of the cows must be slaughtered yearly under ordinary circumstances.

The 6936 slaughtered animals mentioned, according to these figures, would indicate a total of 43,350 cows.

The individual percentage would show the following figures. Of the total number of cows there are annually diseased:

ca. 1.52 per cent. with Chronic udder-streptomycosis.
ca. 0.45 per cent. with Udder-pyobacillosis.
ca. 0.35 per cent. with Udder-tuberculosis.
ca. 0.08 per cent. with Udder-actinomycosis.

ca. 2.40 per cent. of the total number of cows showing udder-infections.

Out of 200 cows one may expect five cases of chronic udder-infection yearly, and of these three are usually streptomycosis and the remaining two may be one of the three other chronic udder-infections. Of these pyobacillosis is the most common and occurs in ca. 1 case in 200 cows; next comes tuberculosis, ca. 1 case in 300 cows, and finally actinomycosis, the least frequent, with ca. 1 case in 1200 cows computed on a yearly basis. Naturally the figures are not so uniform.

The first two figures are fairly constant but the last two are not.

The figures on tuberculosis were taken from our most heavily infected tuberculous districts and are therefore not a fair average for the presence of tuberculosis in the entire country. The figures for the whole country are decidedly lower. In certain parts of Sweden, as in the north, udder-tuberculosis is very rare. Naturally it is not present at all in the districts which are free from reactions.

The figures on actinomycosis taken from the journals of the Veterinary High School of material from the different regions of Sweden are perhaps a fair average for the occurrence of this disease in the country. Actinomycosis, however, is not uniformly distributed in the country. There are regions where udder actinomycosis occurs with relative frequency and others where it is not present at all.

CHAPTER XVI

THE IMPORTANCE OF MASTITIS TO MEAT INSPECTION

Examination

The udder should be examined with an eye for swellings and palpated for thickenings.

When such lesions are observed the half of the udder affected, together with the other half, is laid open by a sagittal longitudinal incision extending through the teats. The appearance of the cut surface facilitates a quick determination of the nature and extent of the condition. When the udder has been thoroughly examined the supra-mammary lymph-glands are inspected. If one or both of these glands are swollen and œdematous one may logically suspect udder-tuberculosis. If this suspicion is not proved to be absolutely unfounded the meat in these cases is judged as though udder-tuberculosis had been diagnosed. When tuberculous foci are present the diagnosis is positive. (I have seen one case of extensive tuberculosis of the supra-mammary lymph-glands, however, without any visible tuberculosis in the corresponding udder. This was a case of wound infection, as a tuberculous wound was found in the gland.) The less frequent actinomycotic foci must not be confused with tuberculosis.

Judgment

Udder.—That half of the udder found to be diseased, together with its corresponding lymph-gland, is unfit for use by man. [The entire udder is condemned.] If a decided sclerosis of an injury is present and no pus can be expressed it may be passed. [Entire udder is condemned.]

If one half of the udder is condemned because of an udder-infection the other half must be critically inspected. It may only be passed when it is manifestly free from pathological lesions and when the udder-infection found in the first half is plainly of a local character (see later). [Entire udder is condemned.]

MEAT.—Only the infectious inflammations of the udder can render the meat unfit for man.

Acute udder-infections are usually local in character. They are milk-infections and wound-infections. The former are the most important and the location of the organisms is in the tubuli, as is the case in the more virulent staphylomycosis with diffuse necrosis of the udder.

Infection of the blood or bacteræmia rarely occurs. It may be recognized by swelling of the spleen, hæmolysis, or by acute embolic pneumonia. A microscopic examination of the spleen or blood must determine the judgment of the meat. If Gram negative rod-shaped bacteria or Gram positive cocci are found the meat is condemned. [Microscopic examination is not necessary. Carcass is condemned on gross lesions of bacteræmia.]

An udder-colibacillosis may cause infection of the meat. This infection is especially difficult to determine. If an acute udder-infection is established and if the cut surface of the udder is severely reddened, a microscopic examination of the juice from the incision in the udder must determine whether or not the meat is fit for food. If a considerable number of fairly large, short and clumpy Gram negative bacilli with rounded ends are found the meat is declared unfit for use by man. [If local the entire udder is condemned. If acute diffused mastitis the entire carcass is condemned. B. A. I. Order 150, Regulation 13, Section 18, d.]

If an acute udder-infection is complicated with gangrene the meat may only be used after we have proved that

it bears no abnormal odor. [The carcass should be carefully examined for lesions of septicæmia.]

Chronic Udder-infections.—Their type plays a great part in the judgment of meat.

Chronic udder-streptomycosis is always of a local character. The auriculo-ventricular valves of the heart may show an endocarditis which might be the cause. Should such really be the cause the meat may be used only after a careful inspection and excision of metastatic lesions in the meat. The heart, together with the organs containing metastatic processes must be condemned. [Entire carcass condemned.]

If gangrene complicates the udder condition the meat must be proved free from odor before it may be used. [Entire carcass condemned.]

Pyobacillosis and actinomycosis of the udder are usually local conditions. They may be generalized, however, the generalization manifesting itself usually in the internal organs only, as the lungs and liver. Condemnation of meat, following generalization of these conditions, is not, as a rule, demanded. In complications of pyobacillosis with gangrene the odor of the meat determines the judgment. [Entire carcass condemned.]

Udder-tuberculosis, as a rule, is an indication of generalized tuberculosis. Udder-tuberculosis is local and primary only when the lungs are free from the disease.

In many cases of generalized tuberculosis only the internal organs are involved, leaving the muscles and bones unaffected, so that all or part of the meat may safely be used. [Entire carcass condemned.] According to the statistics of the abattoir at Malmo for the year 1905, edited by Director A. M. Bergman, 155 cows were affected with udder-tuberculosis of which, in 29 cases, or 19 per cent., the tuberculosis was of slight extent, so that only single parts had to be condemned.

In generalized tuberculosis, therefore, one carefully inspects the body, incises the body lymph-glands and examines the vertebræ and sternum after longitudinal division.

If the udder-tuberculosis is local or the tuberculosis is generalized, but the skeleton and muscles unaffected, and the meat healthy in other respects, the meat can be passed for use. [Entire carcass condemned.]

One should not forget to carefully determine whether or not the supramammary and lumbar lymph-glands are swollen.

Summary

1. Inspection:
   - A. Udder.
   - B. Suprammary lymph-glands.
2. Judgment:
   - A. Udder.
     1. The diseased half of the udder.
     2. The other half of the udder.
   - B. Meat.
     1. Acute udder-infections.
        - Bacteræmia.
        - Colibacillosis.
        - Gangrene.
     2. Chronic udder-infections.
        - *a.* Chronic udder-streptomycosis.
          - Endocarditis.
          - Gangrene.
        - *b.* Udder-pyobacillosis and actinomycosis.
          - Generalization.
          - Gangrene in pyobacillosis.
        - *c.* Udder-tuberculosis.
          - Local udder-tuberculosis.
          - Generalized tuberculosis.
          - Lymph-glands.

CHAPTER XVII

POST-MORTEM REPORT

I. Single Infections

A. STREPTOMYCOSIS

1. *Atypical chronic streptomycosis in both posterior quarters of the udder.*

Both posterior quarters were swollen and firm in consistency. Both quarters presented the same lesions. The incision was very dry. The entire lower portion of the cut surface was even, grayish-yellow and very firm. The lobuli in this region were small and without milk points. Yellow cylinders of fibrin were expressed, and the milk-ducts and cylinders were filled with yellow fibrin. The other areas of the udder showed no lesions.

In the fibrin a few small lancet-shaped Gram positive cocci, arranged as diplococci, were observed (streptococci).

2. *Atypical chronic streptomycosis of the left posterior quarter with nodular necrosis.*

Only the left half of the udder was autopsied.

The left posterior quarter was moderately swollen and firm in consistency.

The incision in the quarter was somewhat dry. The cut surface was even, yellowish-gray and very firm. The lobuli in this region were very small and showed no milk points. Yellow cylinders of fibrin were expressed. Whitish-gray, round, convex, unaltered lobuli showing distinct milk points were distributed here and there. In the posterior inferior part there was a round cavity the size of a hen's egg surrounded by a white, thick connective-tissue wall. It contained yellowish-gray pus and also a grayish-yellow,

firm, necrotic body of irregularly rounded shape and with uneven, ragged external surface. The body was of firm but friable consistency. In the cisterns and the milk-ducts yellow fibrin and viscid croupous membranes were observed. Other parts of the udder manifested no alterations.

A great number of small Gram positive cocci grouped in twos or short chains, for instance, streptococci, were observed in the fibrin and the necrotic tissue. The diagnosis was corroborated by cultures.

3. *Atypical chronic streptomycosis in the right anterior quarter, together with a typical chronic streptomycosis in both posterior quarters.*

The right anterior quarter of the udder was slightly changed, disproportionate and hard. Both posterior quarters were decreased in size, but still thick and somewhat firm.

Right anterior quarter: The incision was somewhat dry. The cut surface was even, grayish-white and firm. The lobuli were small and presented no milk points. Yellow fibrin cylinders were expressed. Groups of grayish-white, convex, soft and elastic unaltered lobuli showing milk points were present here and there. Firm yellow fibrin was observed in the milk-ducts and the cisterns.

Both posterior quarters presented the same lesions. The incision was succulent and the liquid of the incision consisted of a yellow, cream-like pus. The cut surface was even, grayish-white and tendinous. The lobuli were small and presented no milk points. Yellow cream-like pus was found in the milk-ducts and the cisterns. No lesions were found in the other parts of the udder.

In the fibrin of the right anterior quarter a great number of Gram positive cocci arranged in twos or short chains were present (streptococci).

In the pus of both posterior quarters a great number

of Gram positive cocci in long chains were found (streptococci).

B. STAPHYLOMYCOSIS

1. *Staphylomycosis of the right half of the udder as well as the left anterior quarter. Diffuse necrosis of the udder.*

The cow had to be slaughtered.

The right half of the udder as well as the left anterior quarter were greatly swollen and hard.

The lymph-glands were slightly swollen. The incision was succulent with lymph and the cut surface was gray, moist and friable (œdema). The diseased parts of the udder presented the same lesions. The incision was juicy with a dark red, thin fluid. The cut surface was even and diffusely colored red. The consistency was firm and friable, the structure indistinct and no milk points were present. Red fluid was expressed. Here and there dark red-colored, sharply defined parts were observed (blood infiltration). Numerous veins were occluded by firm thrombi. Red-stained fibrin cylinders and a thin fluid were observed in the milk canals and in the cisterns. The loose subcutaneous tissue over the udder was swollen, red stained and of gelatinous consistency. Red fluid was expressed from it. No lesions were found in other parts of the udder.

In the liquid of the incision a great number of Gram positive, uniformly round cocci, arranged in twos or in small bunches, were observed (staphylococci). The diagnosis was corroborated by cultures.

2. *Staphylomycosis in the left half of the udder. Diffuse udder necrosis with gangrene.*

The cow had to be slaughtered.

The left half of the udder was greatly swollen and of firm consistency. The left anterior teat was red and greatly swollen. The corresponding lymph-gland was slightly swollen.

Both left quarters manifested the same lesions, although the lesions in the anterior quarter were more advanced than those in the posterior quarter. The incision was succulent and the juice consisted of a thin red fluid. The cut surface was even, uniformly red stained, and of firm but friable consistency. The structure was indistinct. No milk points were present. Red juice was expressed. Here and there poorly defined grayish-green areas the size of a nut were observed, which had a soft elastic consistency and crepitated when incised (emphysema). Small excised pieces floated in water. These parts presented a putrefactive odor. The milk-ducts and the cisterns contained grayish-red firm but friable fibrin cylinders; the subcutis over the diseased anterior quarter was swollen, red and of a gelatinous consistency. A red fluid was expressed. The connective tissue of the teat of this quarter showed the same lesion (œdema). No lesions were seen in the other parts of the udder.

The fibrin contained a great number of Gram positive cocci arranged in irregular bunches (staphylococci). The diagnosis was corroborated by culture.

In the green parts large Gram positive spore-bearing bacilli were seen (putrefactive bacilli).

C. COLIBACILLOSIS

1. *Colibacillosis in the right posterior quarter.*

Only the right posterior quarter was slightly swollen, and its consistency somewhat firm.

The incision at this point was succulent and the juice consisted of yellow flocculent milk. In the lower part of the cut surface was even, yellowish-gray and somewhat firm. The lobuli showed no milk points and yellow fibrin cylinders were expressed. In the posterior part a somewhat sharply circumscribed area was present which was red stained, of

firm but friable consistency and about the size of an egg.

The lobuli showed the same alterations here as those in the yellowish-gray part. Yellowish fibrin was found in the milk canals and cisterns. Other parts of the udder presented no lesions.

A comparatively great number of clumpy Gram negative bacilli showing rounded ends were found in the fibrin and in scrapings taken from the red parts (colon bacilli). The diagnosis was corroborated by cultures.

D. PYOBACILLOSIS

1. *Pyobacillosis in both posterior quarters.*

Both posterior quarters of the udder were moderately swollen. Their consistency was firm. The lymph-glands were slightly enlarged.

Both quarters showed the same lesions. The incision was covered by thick yellowish-green pus. The lower part of the cut surface was even, grayish-white and firm. The lobuli showed no milk points, but small yellow pus foci. Yellow cylinders of pus were expressed. Yellowish-green pus foci ranging in size from a hemp seed to a bean were observed here and there. They presented a faint, disagreeable odor (not putrefaction) and were surrounded by a white, firm, tough connective-tissue capsule. A few milk-ducts were thickened and formed large irregular cords which, on cross section, presented a yellowish-green centre of pus surrounded by a firm, white connective-tissue capsule, and outside of that by thickened lobuli. Yellow, spongy fibrin was found in the teats and milk-ducts. Other parts of the udder presented no lesions.

A great number of small, thin Gram positive bacilli were found in the yellowish-green pus (pyogenes bacilli).

2. *Pyobacillosis in the right half of the udder as well as in the left posterior quarter. Complication with gangrene.*

The right anterior quarter of the udder was moderately swollen and hard. Both posterior quarters were slightly swollen and firm in their inferior parts.

Right anterior quarter: The incision was smeared by a yellowish-green, thick pus. The cut surface was even, grayish-white, and firm. The lobuli showed no milk points, but yellow pus points instead. Here and there a number of yellowish-green pus foci the size of a hemp seed were present which were surrounded by a thick, white fibrous capsule, the inside of which showed a green pigmentation. These foci gave off a disagreeable putrefactive odor. A few of these pus foci were cylindrical, extended and formed fistulæ. Gray spongy fibrin was present in the milk-ducts and cisterns.

Both posterior quarters presented similar but somewhat less widely distributed lesions. Other parts of the udder showed no alterations.

In the fibrin and yellowish-green pus a large number of small, thin Gram positive bacilli (pyogenes bacilli), as well as other bacteria, were observed (cocci, Gram negative rods, putrefactive bacteria).

E. TUBERCULOSIS

1. *Tuberculosis of the left anterior quarter. Early tuberculosis.*

The left anterior quarter of the udder was greatly swollen and hard.

The corresponding lymph-gland was greatly swollen. The incision was juicy with lymph. The cut surface was gray and showed no caseous foci. Its consistency was soft and friable.

The incision in the diseased quarter of the udder was succulent with milk. The lower part of the cut surface was even, firm, and gray in color. The lowest part of this

area was uniform, and the superior part of it was irregular, nodular or coarsely granular. The lobuli in this region showed no milk points, but here and there, especially in the lower part, a number of small, yellow caseous points or fine caseous streaks (caseous necrosis in the collecting tubules) were present. Yellow creamy fluid was expressed (softened necrosis). The superior and greatest part of the cut surface was strongly convex, whitish-gray and of very soft and elastic consistency. The lobuli of this area were enlarged, strongly convex and showed distinct white milk points. Milk-ducts and cisterns contained milk. Other parts of the udder showed no lesions.

A number of fine acid-fast rods were found in the yellow cream-like fluid (tubercle bacilli).

2. *Tuberculosis in the right posterior quarter. Advanced tuberculosis with calcification.*

The right posterior quarter of the udder was greatly swollen and hard.

The corresponding lymph-gland was greatly enlarged. Its incision was juicy with lymph and the cut surface showed a number of yellow caseous points and yellow branched caseous streaks in the cortex which often showed whitish-yellow calcareous centres.

The incision in the diseased quarter was dry and gritty. Cut surface even and firm. Its superior portion was irregular and nodular. The lobuli of this area showed no milk points, but small yellow caseous points or caseous streaks. Caseous cylinders were expressed. The inferior part of this area was uniform, grayish-yellow and dry. The lobuli of this area presented no milk points, but numerous thickly placed yellow caseous points and caseous streaks. Other lobuli were entirely yellow and showed yellowish-white calcareous centres or calcareous streaks. At numerous points yellow, firm caseous foci showing yellowish-white calcareous streaks were found which were surrounded by a

thin white connective-tissue capsule. Otherwise the udder showed no lesions.

The cheesy foci showed fine acid-fast rods (tubercle bacilli).

3. *Tuberculosis of the left posterior quarter with ulceration through the skin and complication with gangrene. Advanced tuberculosis with softening of the caseous foci.*

The left posterior quarter was greatly swollen and hard. Posterior to and on either side of the teat there were two fistulous openings about the size of a pencil containing a yellow, stinking fluid.

The corresponding lymph-gland was greatly swollen. It was gritty and dry on section (calcium). For the most part the cut surface was yellow, firm and hard, due to necrosis and calcification.

The diseased quarter of the udder was gritty, on section, and was smeared by a yellow caseous mass. The lower part of the cut surface was even, yellowish-gray and uniform. The lobuli showed no milk points, but caseous points were present.

In addition to these, round cavities were seen which ranged in size from a pea to a walnut, and contained a yellow, thick fluid mass of a plaster-of-Paris consistency, due to lime. A thin, white wall of connective tissue surrounded these cavities. Each fistulous opening communicated with an oval or cylindrical cavity the size of a hen's egg. These contained a dark yellow stinking fluid, showing a consistency of plaster of Paris. The walls of the cavity consisted of thick, white, tough connective tissue, the internal surface of which was stained dark bluish-green. The superior and smallest part of the cut surface was convex, whitish-gray, soft and elastic. The lobuli here were large and showed distinct milk points. Between this part and the severely diseased area a yellowish-gray, irregular, granular line of demarcation was visible. A few yellow,

free, croupous membranes were found in the milk-ducts and cisterns. No lesions were found in the other parts of the udder.

Fine acid-fast bacilli were found in the caseous foci (tubercle bacilli).

F. ACTINOMYCOSIS

1. *Actinomycosis of both posterior quarters.*

Both posterior quarters of the udder were slightly swollen. The consistency was firm.

The lymph-glands were not enlarged and on section showed no lesions.

Both posterior quarters showed the same lesions. The incisions were somewhat dry. The lower part of the cut surface was even, gray and irregular. It showed a number of thickly placed yellowish-gray, soft, strongly convex foci (actinomycomata), which ranged in size from a hemp seed to a pea and showed discrete yellowish-brown points. Other parts of the udder manifested no lesion.

Under the microscope brown bodies showed a radiating structure and a distinct peripheral layer of club-shaped structures (actinomyces fungi).

2. *Actinomycosis of the left anterior quarter. Advanced actinomycosis.*

The left anterior quarter of the udder was greatly swollen and hard. The corresponding lymph-gland was moderately swollen and the cut surface showed a number of convex soft yellow points the size of a pea, showing small discrete yellowish points.

An incision in the diseased quarter was dry. The inferior greater part of the cut surface was yellowish-gray, somewhat even, and presented a number of round, soft, convex relaxed foci the size of a walnut or larger, which contained a number of small, discrete, yellowish-white points. The milk-ducts and cisterns contained a yellow flocculent fluid. Their mucous membranes presented

rounded, soft, gray, relaxed, tumorlike growths from the size of a pea to that of a hazelnut, which had grown in through wounds in the mucous membranes. On section these growths manifested small discrete yellowish-white points. Otherwise the udder presented no lesions.

Microscopic examination of scrapings from the soft foci revealed actinomyces colonies showing rather indistinct radiation.

II. Mixed Infections

A. Chronic infections, complicated with acute infections

1. *Pyobacillosis of the left half of the udder, complicated with staphylomycosis in the posterior quarter.*

The cow had to be slaughtered.

The left half of the udder was hard and swollen, the posterior quarter greatly so, and the anterior quarter to a moderate degree.

The corresponding lymph-gland was not swollen and it showed no lesions on section.

The incision in the left posterior quarter was succulent and the juice consisted of a red turbid fluid. The cut surface was even and diffusely reddened. The consistency was firm but friable, and the structure indistinct. The lobuli presented no milk points, but here and there single bright red colored points were seen (pus points). Red fluid and dark gray cylinders of pus were expressed. In other areas a number of round cavities ranging in size from a pinhead to a bean were seen which contained reddish-gray thin fluid or thicker caseous matter. The cavities were surrounded by a thick, white, tough capsule of connective tissue showing a smooth internal surface. A few sharply circumscribed areas were dark red, due to blood infiltration. A few veins were occluded by firm red-stained thrombi.

The entire cut surface of the udder presented a stinking odor. The milk-ducts and cisterns contained a thin fluid and their mucous membranes were red-stained.

The lower portion of the cut surface of the left anterior quarter was reddish-yellow and firm, and the structure distinct. The lobuli showed no milk points, but small yellow pus points were frequent. Cylinders of pus were expressed. Here and there yellowish-green caseous pus foci the size of a pea and surrounded by a connective-tissue capsule were observed. The cut surface presented no odor. The mucous membranes of the milk-ducts were covered by loose, grayish-yellow croupous membranes. Other parts of the udder showed no lesions.

The liquid from the incision in the posterior quarter presented a great number of small thin Gram positive bacilli (pyogenes bacilli), together with a moderate number of Gram positive round cocci arranged singly or in bunches (staphylococci), besides other bacteria (putrefactive bacteria). The diagnosis of the staphylococci was corroborated by cultures.

In the caseous pus of the anterior quarter only small thin Gram positive pyogenes bacilli were found.

B. TWO CHRONIC INFECTIONS

1. *Typical chronic streptomycosis in all four quarters, complicated with tuberculosis in the right posterior quarter.*

The right posterior quarter of the udder was moderately swollen and hard. The other quarters were decreased in size, yet still thick and firm.

The right lymph-gland was enlarged. The incision was succulent with lymph. On cut section the cortex presented small yellow caseous points. The consistency was friable. Incision in the right posterior quarter presented a small quantity of yellow cream-like pus. The cut surface was

even and firm. Its lower part was irregular, nodular and yellowish-gray. Deeply the granules were numerous, but superficially few. The lobuli here showed no milk points, but small yellow caseous points and caseous streaks. The superior part of the cut surface was whitish-gray and firm as tendon. The lobuli showed no milk points in this area. Yellow cream-like pus was expressed.

The other quarters were also purulent when incised. The cut surface was whitish-gray, even, uniform and fibrous in consistency. The lobuli were small and showed no milk points. Yellow cream-like pus was expressed.

Small acid-fast bacilli (tubercle bacilli) were found in the caseous foci.

Gram positive cocci in long chains (streptococci) were found in the yellow cream-like pus.

2. *Pyobacillosis in the left posterior quarter complicated with tuberculosis. Ulceration through the skin and gangrene.*

The left posterior quarter of the udder was greatly swollen and hard. Posterior to the teat a fistulous opening the size of a goose quill was found which discharged yellow stinking pus.

The corresponding lymph-gland was greatly swollen and the incision juicy with lymph. The cut surface presented no caseous points. The consistency was friable.

The incision in the diseased quarter was smeared with a yellow, soft caseous mass. The cut surface was even, firm, yellowish-gray, and showed a number of round cavities ranging in size from a hemp seed to a nut. They contained yellowish-green thick pus and were surrounded by a thick, white connective-tissue wall (abscesses). A cavity the size of two fists was found in the posterior part, which contained yellow, stinking pus, and was surrounded by a fibrous wall, the internal surface of which was dark green in color. This cavity was in communication with the previously mentioned

fistula. Between the abscesses the cut surface was yellowish-gray and irregularly granular. The lobuli of this area showed no milk points, but great numbers of small yellow caseous points or caseous streaks instead. Caseous plugs were expressed. Gray fibrin was observed in the milk-ducts and cisterns. Otherwise the udder presented no lesions.

In the yellowish-green pus from the abscesses numerous small, thin Gram positive bacilli were found (pyogenes bacilli).

In the yellow, cheesy areas thin acid-fast rods were observed (tubercle bacilli).

CHAPTER XVIII

A FEW REPORTS OF CONTAGIOUS UDDER-INFECTIONS

I. Contagious Udder-streptomycosis

A. REVIEW OF A DESCRIPTION BY M. SANDGREN, VETERINARIAN AT YSTAD

During a period of ca. two months, twenty cows were affected with a contagious mastitis. The condition occurred principally in good milking cows. The disease began with intense fever and diminished appetite. It spread rapidly from one quarter to another until the entire udder was soon affected. The diseased quarters were moderately swollen. The quantity of milk was lessened and it was yellow and mixed with pus and flocculi of fibrin.

The sample submitted was almost normal in appearance and of yellowish-white color. The sediment consisted of a moderate amount of yellowish-white pus containing a moderate number of Gram positive streptococci arranged in twos. The diagnosis was corroborated by cultures.

B. REVIEW OF A DESCRIPTION BY S. LINDKVIST, VETERINARIAN AT BROSARP

An obviously contagious mastitis occurred suddenly in a cow stable, and in the course of a few days four cases developed. The stalls of the animals affected were situated in different parts of the stable. The udders were swollen and painful, but not hard. The milk at first was comparatively normal, but became mixed with pus and croupous flakes. The animals showed no fever, but a diminution of appetite. They showed great weakness. They preferred to lie down and could only get up with great difficulty.

The milk sample was of normal appearance and of a grayish-white color. The sediment consisted of a large quantity of yellow pus which contained a moderate number of Gram positive streptococci arranged as diplococci.

C. REVIEW OF A DESCRIPTION BY H. WELIN, VETERINARIAN AT STORA HOF

Contagious mastitis was present in a cow stable for three years, which affected all the cows. The animals manifested a sudden severe swelling and hardness of the udder with purulent milk or a slight swelling of the udder with somewhat normal viscid or flocculent milk. In addition these latter cases often showed swollen and painful tarsal joints, due to metastatic arthritis. One case died, but was not autopsied. In the same stable two similar cases developed. The diseased cows showed fever and weakness, swelling of the udder, together with swollen, painful joints. The animals could scarcely stand, and the quantity of milk was decreased.

The milk sample submitted was nearly normal in appearance. The sediment consisted of a moderate quantity of whitish-yellow pus, containing many streptococci in short chains.

II. Contagious Udder-pyobacillosis

REVIEW OF A DESCRIPTION BY K. KUHLMAN, VETERINARIAN AT LANGHUNDRA

In a cow stable containing approximately sixty cows, six developed contagious mastitis, by degrees, in one month. Usually only one quarter was affected, sometimes two. The diseased quarter was swollen and hard, but not painful. The milk suddenly became flocculent, then purulent, thick and stinking.

The affected cows were isolated and treated with boric

acid injections and iodine embrocations. One cow was slaughtered. The enzoötic disappeared following this treatment. In the same stall similar sporadic cases developed.

The milk sample submitted showed a somewhat normal appearance, but was flocculent. The sediment consisted of a moderate quantity of pus containing numerous small, thin Gram positive bacilli (pyogenes bacilli). The diagnosis was corroborated by autopsy of the udder sent in for examination following slaughter of the animal.

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